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COMMUNICATIONS

THE SIGNIFICANCE OF HEREDITY IN OPHTHALMOLOGY. PRELIMINARY SURVEY OF HEREDITARY EYE DISEASES IN TASMANIA*

BY

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(Concluded)

Optic Atrophy—Hereditary (Leber's)

Although previous mention has been made of this disease in the literature, it was first described in detail with pedigrees, by Leber in 1871, since when considerable research has been done in many countries. Consequently Julia Bell was able in 1931 to publish 237 pedigrees, two of which were recorded by Hogg from Tasmania, and are included—with his permission—with my pedigrees. To Hogg's two pedigrees I am able to add one complete (No. 31) and one very incomplete one (No. 32) of my own. The cause of the incompleteness of No. 32 was the fact that propositus was endeavouring to receive compensation, and until taxed, repudiated all suggestions that his condition was hereditary, which is a further illustration of the difficulties of a pedigree recorder.

I propose to discuss the four Tasmanian pedigrees together under the following headings, but have nothing new to add. So

* Continued from p. 108.

much has been written about Leber's disease, and so little new elucidated that I have found this section of my work the most difficult to write.

Sex Incidence

At once it became apparent that males greatly predominate in all four pedigrees, and the percentage of males affected is given in the following table:—

Pedigree No.	Males	Females	Percentage of Males
10	19	0	100 per cent.
39	3	0	100 per cent.
42	25	2	92·55 per cent.
43	3	1	75 per cent.
Totals	50	3	94·3 per cent.

This is certainly a higher rate than Julia Bell found in European and Japanese pedigrees, which were respectively 84·8 per cent. and 59·1 per cent. of males.

Age of Onset

Hogg, when describing his two pedigrees (Nos. 33 and 34), gives no indication of the age of onset in cases examined by him; and as in my 2 pedigrees only 4 of the cases were examined by me this question with regard to Tasmania cannot be answered definitely. In passing I might mention that in pedigree No. 31 the ages of onset were:—In case III/43 at 27 years, in case IV/7 at 40 years, in case IV/45 at 25 years, in case IV/47 at 20 years, while in pedigree No. 32, in case II/13, the onset was at 19 years.

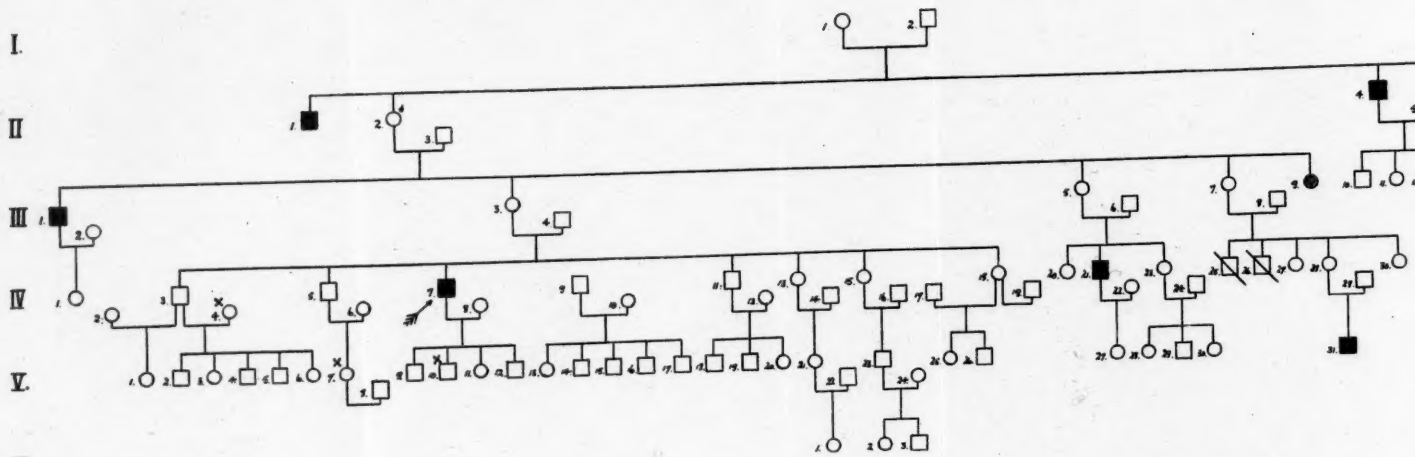
Signs and Symptoms

In three of my cases the onset was sudden, while in the fourth the mode of onset was doubtful. In all four cases both eyes were affected simultaneously, and in all eight cases there were central scotomata and no peripheral constriction. In two of the eight eyes the central scotomata were only partial, and I was able to correct the sight in one eye to 6/24 and in the other eye to 6/12 vision. This shows that the prognosis was not altogether hopeless in two cases out of four.

Causation

In none of the four cases was there any suspicion of pituitary dysfunction, nor were the visual fields obtained, in the slightest degree, suggestive of this condition; and Dr. Hogg reports that

No. 31. LEBER



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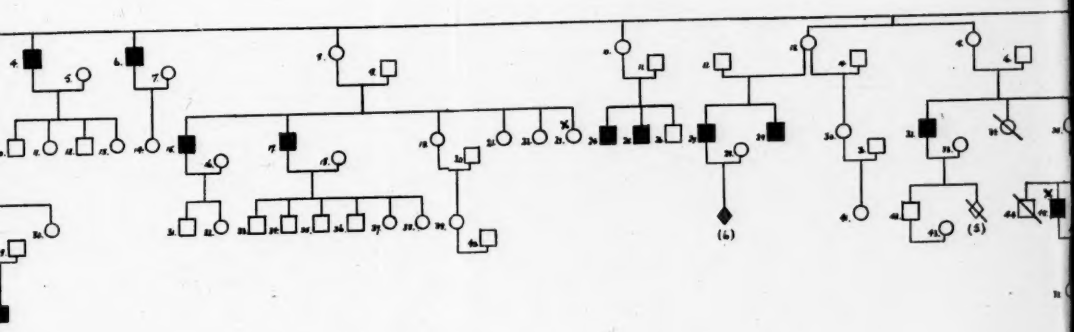
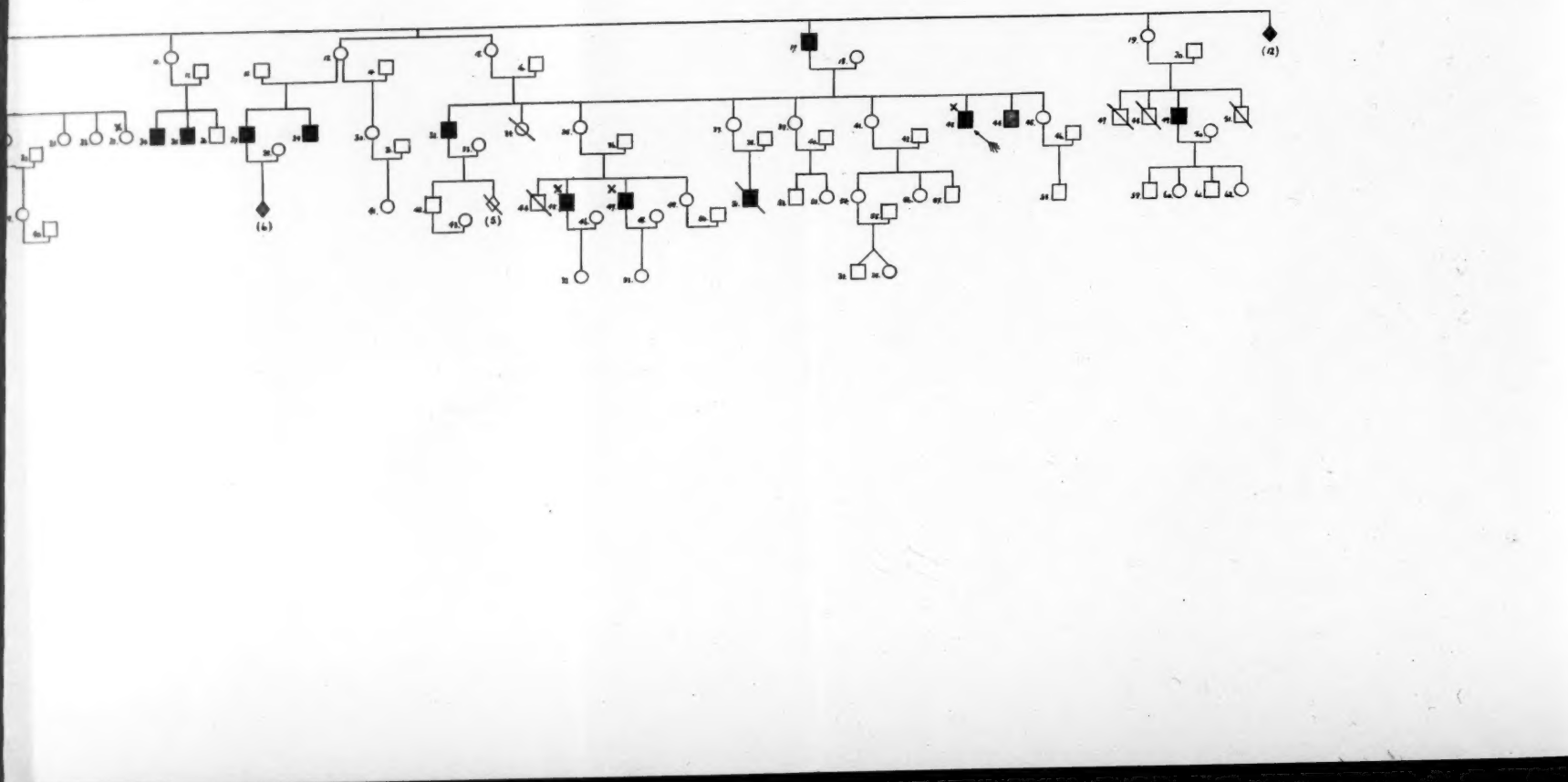
LEBER'S DISEASE.




■ - Affected Males
● - Affected Females
◆ - No History
⊘ - Dead.
× - Examined.
➶ - Propositus.

his X-ray findings of the sella turcica were negative in all cases. Gowers in 1904 declared that the disease was an abiotrophy (Swab, 1934). I feel certain he came nearer the truth than any previous worker, and research during the subsequent 32 years has not been able categorically to contradict his assertion. If Gowers is right —and there seems little doubt that he is—then the correct line of treatment is prophylactic, that is, the prevention of breeding by all carrier females.

Mode of Transmission

In the four Tasmanian pedigrees there is no variation in transmission from those quoted from England and Scotland. In every case the transmission was through an unaffected female who frequently has affected brothers; and in no case did an affected male or female appear to transmit the disease, as was found in the pedigree of congenital optic atrophy reported by Thompson (1935). The disease in Tasmania is therefore of the sex-linked recessive type of Lossen. (Macklin, 1927).

There is no history of consanguinity in any of the four Tasmanian pedigrees, and Julia Bell (1931) records consanguinity in only 18 of her 237 pedigrees; so that this factor does not appear to be a very important one. Nevertheless Russell (1931) reported a pedigree in which a consanguineous marriage appears to have precipitated the disease in three brothers, in an otherwise clean stock, with no affected members in previous generations.

Treatment

Kuhn (1931) reported surgical exposure of the chiasma of a male with symptoms of four weeks' duration, no abnormality was detected, and no visual improvement resulted. A further proof that to date the only available treatment is prophylactic.

Comments on Pedigrees

Pedigree 31.—I consider this pedigree accurate, and fairly complete as it was first given to me by III/43, and later was verified through IV/7. As many of the family are living in New Zealand, and some in Fiji, as well as many on the Continent of Australia, it has been impossible to examine the majority of those affected. V/31, a male who lives in Fiji, and is in his early twenties, is the most recent member to become affected.

III/23. Normal.
III/43. Optic Atrophy. R. and L. R.V.C. correction = 6/12.
IV/7. Optic Atrophy. R. and L. R. and L.V. = <6/60.
IV/45. Optic Atrophy. R. and L. R.V. = 6/24.

Pedigree No. 32.—As explained this pedigree is grossly incomplete, but I hope at a later date to glean more details of further affected members who undoubtedly exist. The one member examined, II/13, had <6/60 R. and L.E.

Pedigrees Nos. 33 *and* 34.—These are published by kind permission of Dr. G. H. Hogg of Launceston, Tasmania; but I am unable to give any details at all. Dr. Hogg in private communication regrets that he has no details available (other than those given when he published the pedigree in the Medical Journal of Australia, on March 24th, 1928), except that the members came from the northern end of this island, and are unlikely to be included in my complete pedigree (No. 31).

REFERENCES

BEDELL, A. J. (1934).—*Amer. Jl. Ophthal.*, Vol. XVII, No. 3, p. 195.
BELL, J. (1931).—Treasury of Human Inheritance. Vol. II, Part IV. Cambridge.
CORDES, F. C. (1933).—*Trans. Amer. Ophthal. Soc.*, Vol. XXXI, p. 289.
HOGG, G. H. (1928).—*Med. Jl. Austral.*, March 24, p. 372.
HOGG, G. H. (1936.—Personal Communication.
KUHN, A. S. (1931).—*Arch. of Ophthal.*, March. (Abstract.)
MACKLIN, M. T. (1927).—*Canad. Med. Assoc. Jl.*, Vol. XVII, p. 1339
RUSSELL, W. R. (1931).—*Trans. Ophthal. Soc. U.K.*, Vol. LI, p. 187.
SWAB, C. M. (1934).—*Nebraska Med. Jl.*, Vol. XIX, p 184.
THOMPSON, A. H. (1935).—*Proc. Roy. Soc. Med.*, Vol. XXVIII, p. 1415.

Pterygium

Tasmania, which is famous throughout the Antipodes for its delightful climate, is practically free from progressive pterygium, trachoma, hyperkeratosis and rodent ulcer of the lids. During the last 5½ years out of 4,880 private case records I find the following statistics:—

Pterygia	44 cases = 0·9	per cent.
Trachoma (old)	4 cases = 0·08	per cent.
Trachoma (active)	4 cases = 0·08	per cent.
Rodent ulcer of lids	5 cases = 0·1	per cent.

Of these 44 cases of pterygium—10 cases have had to be dealt with surgically, so that it is evident that pterygium *per se* is a disease of little consequence in Tasmania, although on the Continent of Australia it is a constant source of concern.

Of these 44 cases I can find an hereditary influence in only 2 cases—2 brothers whose maternal parent suffered from the same condition.

Sex Incidence

Reference to hereditary pterygium in the literature is very scanty. Macklin (1927) reports two pedigrees from the literature

—one of 3 affected males and 5 affected females in 4 generations; and another of 10 affected males and 2 affected females in 6 generations.

My pedigree consists of two males and one female, and this predominance of males on the whole is possibly influenced by greater outdoor activities of males.

Mode of Transmission

Macklin considers the transmission as dominant, being transmitted by both parents to offsprings of either sex.

Comments on Pedigrees

Pedigree No. 35.—I/2 is dead, but definitely had a pterygium which her sons think was on her right eye.

II/1 has a right active pterygium as also had II/2. I marsupialised both after the McReynold's method, with satisfactory result.

REFERENCES

GATES, R. R. (1929).—Heredity in Man. London.
MACKLIN, M. T. (1927).—*Canad. Med. Assoc. Jl.*, Vol. XVII, p. 698.

Hereditary Ptosis

In a limited pedigree such as the one reported by me, the author should be extremely careful that the described condition is not due to birth injury. In this pedigree I certainly cannot be dogmatic, but as the uncle of the patient examined by me has definitely a bilateral ptosis, it is strongly suggestive that the condition is hereditary in nature. On the other hand, first the fact that birth injuries to the eye are prevalent in Tasmania (and I hope to report a series shortly), secondly that Briggs (1919), Usher (1925) and Macklin (1927) all assert that ptosis is dominant in transmission (which is not so in this pedigree), and thirdly, that in my pedigree only males are affected (see sex incidence), make me somewhat doubt the authenticity of the family record. Nevertheless the propositus volunteered the facts at the first examination of his son, and I have been able to confirm his assertions in one instance at least.

Sex Incidence

According to Macklin (1927), the sex incidence is about equal taking all reported pedigrees collectively, but in my pedigrees only males have been affected to date.

Mode of Transmission

In at least five of the authors referred to, the mode of transmission has been dominant, but my pedigree does not confirm this.

Associated Defects

Epicanthus.—Usher (1925) and Clausen (1923) surveyed the literature on hereditary epicanthus with ptosis—they found them frequently reported together, and although ptosis occurs frequently without epicanthus, yet they could only find one instance of hereditary epicanthus *per se.*

High Refractive Error, Nystagmus, and Paralysis of Superior Rectus.—These also have been frequently reported in pedigrees of hereditary ptosis. Usher (1925) reports that in 18 eyes examined, only two had normal vision. My examined case had high refractive error, and affected superior recti.

Squint with Amblyopia ex Anopsia.—This likewise is a fairly frequent concurrent sign, and was present in my case, in the form of a convergent strabismus of 20° and partial amblyopia in his left eye.

Pathology

According to Briggs (1919), the defective action of the levator may be due to five causes:—

a Defective development.
b Adhesion to superior rectus.
c Abnormal insertion.
d Replacement by connective tissue.
e Entire absence.

Treatment

In view of this pathology, to attempt advancement of the levator appears to be futile, and therefore some other muscle must be attached to the lid, and used to elevate it. This can be carried out by utilising the superior rectus as in the classical Motais operation, or the more recent operation of Greeves (1933), or by utilising the frontales by the classical operation of Hess.

Comment on Pedigree

Pedigree No. 36.—II/1. Male. Not examined or confirmed.
III/8. Female. Examined. No ptosis.
III/7. Male. Not examined but confirmed. Patient has only a slight degree of bilateral ptosis.
IV/3. Male. Examined. No ptosis.

IV/4. Female. Examined. No ptosis.

IV/18. Male. Not examined or confirmed.

V/1. Male, aged 5½ years. Marked bilateral ptosis with head tilting, and contraction of both frontales. Marked by defective movement of right globe up, and up and out, and slight defective movement of left globe up, and up and in. Left convergent concomitant strabismus of 20° with 6 dioptres of left hyperphoria.

An interesting point in this child is this:—Having corrected horizontal muscle error by exercises and operation, I attempted to correct his vertical error with 6 prism dioptres, combined with his hypermetropic lenses. The wearing of this prism increased his vertical error, so that without the prism this stood at 12 prism dioptres, while with it the vertical error was stationary at 6 prism dioptres. Apparently he has an abnormal vertical correspondence of 6 prism dioptres, an unusual but not unreported condition.

REFERENCES

BRIGGS, H. H. (1919).—*Amer. Jl. Ophthal.*, Vol. II, p. 408.

CLAUSEN, W. (1923).—*Zentralbl. f. die ges. Ophthal. und ihre Grenzgebiete*, Vol. XI, No. 11, p. 481.

MACKLIN, M. T. (1927).—*Canad. Med. Assoc. Jl.*, Vol. XVII, p. 55.

McILROY, J. H. (1929).—*Proc. Roy. Soc. Med.*, Vol. XXIII, p. 285.

USHER, C. H. (1925).—*Ann. Eugenics*, Vol. I, p. 128.

USHER, C. H. (1935).—*Trans. Ophthal. Soc. U.K.*, Vol. LV, p. 164.

TREATMENT

GREEVES, R. A. (1933).—*Brit. Jl. Ophthal.*, Vol. XVII, No. 12, p. 741.

Retinal Detachment

While perusing the available literature, it became apparent that much confusion exists in medical circles regarding the terms familial and hereditary. According to Gould's Medical Dictionary, 4th Edition, the meaning of these two words is as follows:—

Familial.—Characteristic of a family.

Hereditary.—Transmitted from parent to offspring.

I must at once admit guilt myself in including in this survey at least two pedigrees which are only familial, and not hereditary in nature; but I plead privilege in so much as this is only a preliminary survey, and I hope in a final survey to prove their hereditary nature.

The Librarian of the Royal Society of Medicine has been able to secure reports of six pedigrees of hereditary detachments of the retina for me, but one of these is purely familial in character, and therefore not pertinent to the subject under discussion. Furthermore, five of the pedigrees are in myopic families, so that really these pedigrees should be under the heading of—myopia with detachment as a concurrent complication, which conforms to the classification of other myopic defects, such as posterior staphylomata. These points are strongly exemplified in

Schmelzer's (1929) pedigree, in which both parents, and every member of the only affected sibship were myopic, and 3 of the 5 sibs developed detachment, although neither parent had the complication. Again in Bogatsch's (1911) pedigree of 38 members of a family, 11 had myopia, and of these 11, 4 had bilateral detachments, and three unilateral detachments. The one non-myopic hereditary detachment reported, is that described by Pagenstecher in 1913 and reported in the Graefe-Saemisch-Hess Handbuch (1916). Here a grandfather and two grandsons suffered from detachments, with evidence of hypermetropia only. The last-mentioned pedigree bears a definite similarity to the pedigree I am about to present and discuss, in so much that the detachments were definitely hereditary, and non-myopic in nature.

Arruga (1933) has clearly pointed out in his paper the rarity of the hereditary factor in detachments in Spain; and the pedigree he quotes of a father, two sons and a daughter is the most extensive pedigree he found in the examination of 682 cases of detachment of the retina between 1910 and 1932. Anderson (1931) in his monograph dismisses the influence of heredity in half a page.

Treatment

Macklin (1927) quotes all authors as describing the mode of transmission as dominant. Treacher Collins described it as recessive, and Pagenstecher found it a sex-linked recessive character. Macklin explains this disparity as possibly due to detachment of the retina, being a defect associated with several eye diseases. I think this is a most probable explanation.

Comments on Pedigrees

Pedigree No. 37.—The first member of this pedigree to come under observation five years ago, was III/6. He was then 11 years of age and had a complete right retinal detachment, with a hole at 7.30 o'clock, 2 D.D. from the ora. On this eye he had had three Gonin operations performed elsewhere, without success. The left eye had a complicated cataract obscuring all fundus details, and in this eye also was a partial aniridia. By partial I mean an almost complete aniridia of the mesoderm of the iris, with loss of half the ectoderm. The condition on inspection was from centre outwards:—A very dilated pupil, wide ring of iris pigment followed by narrow ring of iris stroma. (See Figure 1.)

Four years later I had to see this boy again for pension purposes, and he then had a mature complicated cataract in his right eye, obscuring all details, but his left eye was most enlightening. The left lens had become shrunken and dislocated upwards revealing a complete detachment of the retina which was white, opaque

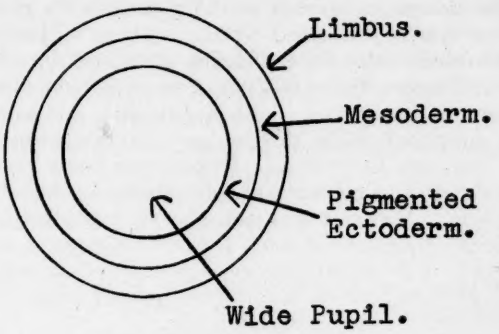


FIG. 1.

and studded with glistening crystalline deposits. The detachment in this eye resembled very much the bilateral detachment of III/1, about to be described. The right vision was now doubtful perception of light, and with the left eye no perception of light was obtainable.

The same year I saw III/1, aged 5 years, with bilateral detachments of each retina which were semi-solid in nature, *i.e.*, pseudogliomatous in type. At that time I had no idea she was a cousin of III/6. There were gross vitreous opacities in the right eye, but no hole in either retina. Three years later I saw this case again as an inmate of the Blind Institution. The right eye now had a complete detachment, while the left detachment was unaltered. In the upper half of the left retina was gross choroidal scarring but no hole was discernible in this eye either. The vision in her right eye was now counting fingers at 6 inches, and in the left eye counting fingers at 3 feet.

Case III/8, aged 11 years, came under observation last year. His left sight failed two years previously, and had been treated by the family doctor who can give me no details of his past history. Later he was seen by Dr. Hogg (1936) who found his eye in the same condition as that about to be described. When I examined him his right eye was normal, but took a correction of $\frac{+3{\cdot}0 \text{ D.S.}}{+0{\cdot}5 \text{ D.C.} \rightarrow 180^\circ}$ which gave 6/6 and J.1 vision. His left eye had a mature cataract with perception of light but no projection. I therefore can only conjecture that a detachment is present in this eye; and if this is so, the high hypermetropia in the right eye is an interesting factor.

Case III/6 and III/8 had a negative W.R. but III/1 had a doubtful reaction at first, which rapidly became negative with

antispecific treatment, so that syphilis is certainly not the causal factor; and this is confirmed by the authors of pedigrees given in the references. But I feel that the unilateral aniridia of III/6, which according to Bell (1932) is a most uncommon condition *per se* may be suggestive. Such aniridia, according to Croll (1929) is supposed to be due to late differentiation of the lens delaying true iris formation. In this eye there was a definite lack of associated developmental determinant in the iris mesoderm and ectoderm. May not this lack also be exhibited in the retina with deficient cohesion between the retinal layers, and ultimate detachment? I think this theory is worthy of consideration, and should other members of this family become affected, I may be able to offer further substantiation.

I am unable to find any members with aniridia in the other pedigrees of hereditary detachment studied and quoted in the references, nor does Bell (1932) find detachment associated with any of the 118 cases of aniridia which she has analysed, but Clausen (1923) mentions that it does occur.

REFERENCES

ANIRIDIA

BELL, J. (1932).—Treasury of Human Inheritance. Vol. II, Part V. Cambridge.
CROLL, J. J. (1929).—*Archiv. of Ophthal.*, Vol. II, No. 6, p. 699.
MACKLIN, M. T (1927).—*Canad. Med. Assoc. Jl.*, Vol. XVII, p. 937.

DETACHMENT OF RETINA

ANDERSON, J. R. (1931).—Detachment of the Retina. Cambridge.
ARRUGA, H. (1933).—XIV Concilium Ophthal., 1933. Hispana, T. 11/1 (abstract).
BOGATSCH, G. (1911).—*Klin. Monatsbl. f. Augenheilk.*, Vol. XLIX, p. 431.
CLAUSEN, W. (1923).—*Zentralbl. f. die ges. Ophthal. und ihre Grenzgebiete*, Vol. XI, No. 11, p. 481.
CARTER, J. L. R. (1936).—Personal Communication.
GRAEFE-SAEMISCH-HESS (1916).—*Handb. d. ges. Augenheilk.* 2nd Edition. Bd. VII/2, Kap. X.a/2.
HOGG, G. H. (1936). Personal Communication.
MANN, I. C. (1928).—Development of the Human Eye. London.
MARSHALL, J C. (1936).—Detachment of the Retina. London.
MACKLIN, M. J. (1927).—*Canad. Med. Assoc. Jl.*, Vol. XVII. p. 1336.
SALZMANN, M. (1921).—*Wien. Med. Wochenschr.*, Vol. LXXI, p. 1082.
SCHMELZER, H. (1929).—*Arch f. Augenheilk.*, Vol. C-CI, p. 268.

Retinitis Pigmentosa

It is obvious that the elucidation of the nature of retinitis pigmentosa immediately succeeded the invention of the ophthalmoscope in 1851, for while the symptoms were somewhat typical, the actual diagnosis rested with the fundus examination. In 1853 the first case was described by van Trigh, at Utrecht, and about the same time von Graefe described a case in Berlin, but the credit

of classifying all available information must remain with Nettleship. (Bell, 1922).

Under the above heading Nettleship included the following 6 diseases as belonging to one group of hereditary defects of sight:

(1) Retinitis pigmentosa.
(2) Retinitis pigmentosa sine pigmento.
(3) Retinitis punctata albescens.
(4) Gyrate atrophy of the choroid and retina.
(5) Stationary night blindness.
(6) Choroideremia.

Julia Bell (1922) finds no inclination to depart from this classification, and gives her reasons, while Usher in his Bowman Lecture (1935), also gives ample pedigrees to substantiate this contention.

In my investigation of hereditary eye diseases in Tasmania during the past 5½ years I have been able to find only 14 fresh cases of retinitis pigmentosa, eleven of which I have been able to examine. In not one case could I find an hereditary history, but in seven cases—2 in each of 2 families and 3 in another—there was a familial history, and these are depicted in pedigrees 38, 39, 40. In one family (pedigree 38) the parents were first cousins, and I was able to examine only the female member as the male member refused to be submitted to any examination. The female member in this family also refused all treatment except change of glasses.

Concurrent Anomalies

Ring scotoma.—In none of the 22 eyes examined was there a complete ring scotoma, but in three eyes there was a suggestion of one, though nothing more.

Wassermann reaction.—This was done in 6 cases out of 11, but all 6 tests were negative. One of these 6 cases had a Kahn test done and another of the 6 a Kline test, but both of these were negative also.

Deafness.—Two out of 11 patients complained of deafness—these both occurred in pedigree 39. The elder sister was examined by Dr. Hiller (1933) who reported a bilateral nerve type of deafness.

Obesity, hypogenitalism, polydactylism, or mental diseases (Laurence-Moon-Biedl-Syndrome)—Sorsby (1932)—Savin (1935)—did not appear in any of the 11 cases, neither was there evidence of hyperactivity of the sympathetic system such as Raynaud's disease or chilblains. In view of the number of new pedigrees of this syndrome, reported since Sorsby's article on Laurence in 1932, I have asked my medical and surgical colleagues in Tasmania to draw my attention to any cases they may see.

Pathological Examination

None of the 22 eyes examined has been submitted to pathological examination, but I feel that little new would have been revealed by such examination, as the signs and symptoms of the disease conform very closely those reported from England, and those seen by the author while in London. Therefore I have nothing to add to the theories of causation of this disease, except that the theory of vaso-constriction of the retinal vessels does not appear to hold good in the light of recent surgical treatment. (See treatment.)

Consanguinity

This was apparent in only 2 cases out of 14, *i.e.*, 14·28 per cent. compared with Julia Bell's (1922) estimation of 27·2 per cent. in 817 cases, but I shall say more of this under economic significance.

Sex Incidence

Of the 14 cases discovered no less than 10 were females, giving an incidence of 71·35 per cent., but Bell (1922) found a predominance of males in her series. Dickson and Mitchell (1927) report a predominance of females, but quote no figures or references to substantiate this.

Mode of Transmission

Usher (1935) in his Bowman Lecture describes retinitis pigmentosa as a "Mendelian Recessive through a few large pedigrees with many affected individuals showing predominance." He also gives a pedigree of sex-linked retinitis pigmentosa. My 14 cases show no evidence whatever of being hereditary—all pedigrees given (38, 39, 40) being familial in type. Dr. G. H. Hogg (1936) of Launceston, Tasmania, in a personal communication says that he is of the opinion that there are several cases of retinitis pigmentosa related to one another in Northern Tasmania, but that he has not tabulated them to date, and is unable to give me details.

Economic Significance

On closely examining the first 250 pedigrees of this disease given by Julia Bell (1932), I find the following facts:—

Consanguinity. 95 pedigrees.
No consanguinity. 102 pedigrees.
No record of consanguinity. 53 pedigrees.
Total of accurate records. 197 pedigrees.
Percentage of consanguinity in 197 accurate records = 48·5 per cent.

And so it appears that in 197 pedigrees 48·5 per cent. could have been prevented by stricter laws controlling consanguinity. As at the present time there is no dependable treatment for this progressive and disabling malady, the obvious mode of prevention lies in revised legislation, and it behoves all Ophthalmological Societies to bring this fact to the attention of their respective State Governments, and urge action.

J. B. S. Haldane (1936) quotes Usher's cases when asserting that 27 per cent. of retinitis pigmentosa cases are due to consanguineous marriages; and even at this figure legislation is urgently required.

Education

This should be pursued along normal lines with regular ophthalmological examinations (Report of the Committee on Partially Sighted Children, 1934), until such time as the ophthalmic surgeon considers admission to a sight-saving class or blind institution advisable. But the above report stresses the necessity of repeated and close observation of the vision, fields and fundi of each case. From the onset these patients should never be allowed abroad alone between sunset and sunrise, as their disability renders them unsafe in busy traffic at night.

Treatment

Barrett (1934) and Royle (1930 and 1932) both report retardation of this disease from cervical sympathectomy, while Meighan (1935) and Walch (1935) each report three failures from the same operation. In the discussion which followed this paper by Meighan, Hepburn expressed the opinion that early cases might derive more benefit from the operation—a point stressed by Barrett (1934) and Kerr (1935). Recently I have had 1 patient in this series of 14, operated on the right side only. She is a very early case, aged 10 years, with an incomplete ring scotoma in the right field. I watched the case for three years to confirm the fact that it was progressive, and when the pigment commenced to take on a definite bone-corpuscle formation, and when the field at the same time commenced to show early scotomata, I advised operation. The operation has produced a right Horner's syndrome, but I cannot detect any increase in the calibre of her right retinal vessels nor could Walch (1935) in his three cases. After six months both fields of vision show progressive deterioration, and both fundi increase in pigmentation. We must now patiently await results, in my case, but I fear they will be disappointing.

To date I have not tried acetylcholine on any patient with retinitis pigmentosa.

Allied Diseases

With regard to the other diseases of this group, namely, retinitis pigmentosa sine pigmento, retinitis punctata albescens, gyrate atrophy of choroid and retina, stationary night blindness, and choroideremia, no cases of these diseases have come under my notice in Tasmania, neither—to my knowledge—have any cases been reported. Nevertheless this is only to be expected, as in the British Isles with a population 200 times that of Tasmania, cases of these diseases are of very rare occurrence. (Milner, 1932.)

Comments on Pedigrees

Pedigree No. 38.—II/1. Typical case with temporal island in right field, and moderate myopic astigmatism. Parents were first cousins.

Pedigree No. 39.—III/1. Fields reduced to fixation. Complicated cataracts. Vision down to hand movements. Nerve deafness.

III/4. Sister of the above. Fields reduced to fixation. Left eye still 6/18 with myopic astigmatism correction.

IV/6. Examined for myopic astigmatism, but not affected with retinitis pigmentosa.

Pedigree No. 40.—III/1. Not examined. Affected but no details.

III/5. Fields reduced to 10° right and left. Complicated cataracts. Right and left vision with correction = 6/12.

III/6. Affected, but now dead.

Pedigree No. 41.—This pedigree Dr. Carter of Launceston was good enough to send me, but it did not arrive until after this section of my paper had been completed. Every member has been examined by him and his comments are as follows:—

I/1, aged 46 years. Normal.

I/2, aged 44 years. A typical variety of retinitis pigmentosa. Slight field changes.

II/1, aged 21 years. Typical case.

II/2. Normal.

II/3, aged 16 years. Very advanced case. Fields reduced to 10° from fixation.

II/4. Normal.

II/5, aged 5 years. Typical advanced case. Fields contracted to 30° from fixation.

II/6. Normal.

At the moment he is investigating other branches of the mother's family for evidence of the disease.

REFERENCES

BEDDELL, A. J. (1936).—*Brit. Med. Jl.*, No. 3944, p. 296.
BELL, J. (1922).—Treasury of Human Inheritance. Vol. II, Part I. Cambridge.
CARTER, J. L. R. (1936).—Personal Communication. Launceston.
CRAWLEY, R. H. (1934).—Report of Committee of Enquiry into Problems of Partially Sighted Children.
DICKSON and MITCHELL (1927).—Supplement to *Med. Jl. Austral.*, December 3 p. 462.
HALDANE (1936).—*Brit. Med. Jl.*, February 8, p. 281.
HILLER, B. (1933).—Personal Communication.
HOGG, G. H. (1936).—Personal Communication.
MACKLIN, M. T. (1927).—*Canad. Med. Assoc. Jl.*, Vol. XVII, p. 1341.
MILNER, J. G. (1932).—*Brit. Jl. Ophthal.*, Vol. XVI, p. 418.
USHER (1935).—*Trans. Ophthal. Soc. U.K., Vol. LV, p. 164.*
WHISHAW, R. (1936).—Personal Communication.

TREATMENT

BARRETT, J. (1934).—*Trans. Austral. Med. Congress*, p. 206.
CAMPBELL, GLEN. (1931).—Personal Communication. Vancouver, Canada.
KERR, H. H. (1935).—*Amer. Jl. of Surgery*, Vol. XXVIII, p. 364 (abstract).
MEIGHAN, S. S. (1931).—*Trans. Ophthal. Soc. U.K.*, Vol. LI, p. 124.
MEIGHAN, S. S. (1935).—*Trans. Ophthal. Soc. U.K.*, Vol. LV, p. 93.
ROYLE, N. D. (1930).—*Med. Jl. Austral.*, September 13, p. 364.
ROYLE, N. D. (1932).—*Med. Jl. Austral.*, July 23, p. 111.
WALCH, F. B. (1935).—*Arch. of Ophthal.*, Vol. XIV, p. 699.

LAURENCE MOON-BIEDL SYNDROME

PATON, L. (1936).—*Proc. Roy. Soc. Med.*, Vol. XXIX, p. 751.
SAVIN, L. H. (1935).—*Brit. Jl. Ophthal.*, Vol. XIX, p. 597.
SORSBY, A. (1932).—*Brit. Jl. Ophthal.*, Vol. XVI, p. 727.

Strabismus

There is no doubt that strabismus stands pre-eminent as associated with monocular blindness, as the result of loss of binocular fusion. The loss of monocular vision in itself is not so very devastating nor is the loss of binocular fusion, but with only one functioning eye all squinters are in a most precarious position, and any injury or disease to the non-squinting eye may lead to blindness. To the lay mind the cosmetic effect is paramount but to present day ophthalmologists the acquired amblyopia is the main issue, or at least it should be. To Worth (1929) we must do honour, for he constantly urged that permanent amblyopia ex anopsia could be avoided if taken in hand before the patient reached the age of seven. Gradually the old practice of allowing the squinters to grow out of their squints has passed away, and parents are now bringing their children to oculists at a much earlier age. Even in the past 5 years a great awakening has occurred in the parents of Tasmania, so that some bring their children for advice before they are twelve months old. I feel that this has been largely brought about by the introduction of orthoptic exercises, for as Parsons (1936) says, " It (orthoptic training) has one overwhelming argument in its favour, viz., that

when it is successful it cures the squint." Pugh (1936) and Travers (1936) substantiate this view. Most parents are loath to submit their young offspring to operations, but will submit them to orthoptic training almost indefinitely if they observe even slow progress. It therefore appears to me that amblyopia ex anopsia in adults will have almost vanished in the next generation, and thus another milestone in the prevention of blindness will have been passed.

Hereditary Factor and Mode of Transmission

With the increase in patients seeking early advice, the hereditary nature of strabismus has become more apparent (Mayou, 1935), and in many families a history of cousins, etc., suffering from the same complaint can be frequently elicited. In practically every instance the mode of transmission is recessive, nor does there appear to be any sex linkage. Czelletzer quoted by Travers (1936) asserts that the transmission depends on two recessive Mendelian factors. Worth (1929) found amongst 1,373 cases of squint a history of squint in either parent, grandparent, brother or sister of the patient in no less than 711, *i.e.*, about 51 per cent., while Franceschetti (1930) believes that heredity plays but a small part in the causation of concomitant squint. In Tasmania I should roughly estimate the percentage to be somewhat lower than Worth's figures, but I have no statistics to substantiate this view, as heredity not being a potent factor in the individual diagnosis, had not been inquired into by me in every case before I commenced this paper.

Sex Incidence

There seems to be a preponderance of females in both the heterotropias and heterophorias in my pedigrees. In the cases of hereditary heterotropias I find 21 females and 11 males, *i.e.*, 65·6 per cent., and 34·4 per cent. respectively. While in my cases of hereditary heterophorias I find 5 females and 3 males, *i.e.*, 62·5 per cent. and 37·5 per cent. respectively. In the available literature I am unable to find any reference to this factor.

Unilateral Incidence

From my reported pedigrees of heterotropia it would appear that it is usual for the inherited defect to be transmitted to the same sided eye from the parents to sibs, and although I shall give no details of refractive errors it is remarkable how constantly these are transmitted also.

Comments on Pedigrees

Heterophorias

Pedigree No. 42.—The two affected members are mother (I/2) and daughter (II/2). Each suffered from high hypermetropia, the maternal refractive error being the higher. Each has also marked esophoria—the daughter's being greater for distance, while the mother's error was more marked for near vision—a fact to be expected considering the mother was almost at the age of presbyopia.

Pedigree No. 43.—Here the whole family—mother, father, son and daughter have been examined. The mother's examination was negative, the father showed high hypermetropic astigmatism with 2 prism dioptres of right hyperphoria, and 2 prism dioptres of exophoria, the elder child (the son), had no refractive error, but up to 18 prism dioptres of right hyperphoria on fatigue; while the daughter showed moderate hypermetropic astigmatism, with a right convergent concomitant strabismus with amblyopia ex anopsia.

Pedigree No. 44.—In this pedigree a mother (I/2) with moderate hypermetropic astigmatism had from 4-8 prism dioptres of esophoria, while the youngest daughter (II/3) who was slightly myopic has such a high exophoria that it is not accurately measurable by ordinary tests. With orthoptic exercises this was gradually reduced to 14-18 prism dioptres, and finally she became absolutely orthophoric for near and distance.

Pedigree No. 45.—Here a father (I/1) and daughter (II/3) have marked exophoria. The father has no refractive error of consequence, but has from 7-12 prism dioptres of exophoria. The daughter is hypermetropic but had 18-22 prism dioptres of exophoria both for distance and near, and 4 prism dioptres of right hyperphoria for distance. With orthoptic treatment the exophoria has been reduced to 10 prism dioptres for distance and 1 prism dioptre for near, while the hyperphoria has been reduced to 1 prism dioptre. The mother (I/2) is believed also to have some muscle error but to date has not been examined.

It will be noted in the foregoing four pedigrees that the parental muscle error was less in every case than that of the offspring.

Heterotropias

Pedigree No. 46.—Here the mother (I/1) and daughter (II/1) have amblyopic eyes. (I/1) has grown out of her squint, but has marked esophoria with a cover test. While II/1 with 65° of concomitant convergent strabismus has been successfully operated on by me.

Pedigree No. 47.—There is a history of consanguinity in this

family, and also of squint on both maternal and paternal sides of patient (IV/1) who has congenital amblyopia in the right eye. At least one paternal uncle is amblyopic also, but of course this may be ex anopsia and not congenital in origin. II/5, II/6, III/31 and IV/10 have also been examined. II/5 has hypermetropic astigmatism, but no squint; II/6 has senile cataract with hypermetropia; III/31 has right concomitant convergent strabismus, with high hypermetropia and amblyopia ex anopsia; while IV/10, aged 5 years, has left concomitant convergent strabismus, with amblyopia ex anopsia now under treatment. The left sidedness of the strabismus of IV/10 can be accounted for by a birth injury to that side of her face. The other two squinting members of this pedigree who were examined (IV/1 and IV/10) have right-sided squints.

Pedigree No. 48.—This records two sisters and a distant cousin with squint. IV/1 has been under treatment since 1917, and was first examined by me in 1931 with 15° right concomitant convergent strabismus with correction and high hypermetropia and right amblyopia ex anopsia. In 1936 she had 7° right concomitant convergent strabismus, and 6/36 and J.16 vision with correction. After a course of synoptophore exercises this angle was reduced to 4° and the right vision improved to 6/24 and J.8 with correction. Her sister (IV/4) has been under treatment since 1928, *i.e.*, since 6 years of age. She has also straight visual axes, and 6/9 and J.2 vision with correction in the eye which previously squinted. In this pedigree both are right-sided squints.

Pedigree No. 49.—As only IV/2 has been examined no comments are permissible.

Pedigree No. 50.—Here again squinting is apparent both on the maternal and paternal side of the two examined children (III/10 and III/11). III/10 came under treatment when 8 years of age, with a high hypermetropic astigmatism, 45° left concomitant convergent strabismus and left amblyopia which would not respond to prolonged occlusion. Her post-operative result supplemented with orthoptic exercises was perfect. Her brother III/11, had extremely high pure hypermetropia with 55° concomitant convergent strabismus of alternating type. Despite his abnormal correspondence, after two operations he had a perfect cosmetic result. The fusional result it need hardly be said was poor.

Pedigree No. 51.—In this pedigree six members have been examined. I/2 has low grade hypermetropia, but no squint. II/3 has hypermetropic astigmatism and 2 prism dioptres exophoria for distance, while II/5—her sister who is dead—is reputed to have had crossed eyes. II/6 has high mixed astigmatism but no squint.

III/1, the daughter of II/3, has high hypermetropic astigmatism, 35° left concomitant convergent strabismus and left amblyopia ex anopsia. Occlusion followed by exercises and operation have restored both visual axes and vision to normal. III/4 has high hypermetropic astigmatism alone, while III/5 has right concomitant convergent strabismus, high hypermetropic astigmatism, and amblyopia ex anopsia. Both are right-sided squints.

Pedigree No. 52.—The paternal responsibility for strabismus is here clearly shown. Only IV/1 and IV/2 has been examined, and both are under early treatment. Both have squints and high hypermetropia and amblyopia ex anopsia in the left eye, and the onset in each was under 2 years of age.

Pedigree No. 53.—I have only examined two members of this pedigree, neither of whom has a squint, but the propositus III/9 assures me that every squinting member of this pedigree, except II/10, squints with the right eye.

REFERENCES

CANTONNET, A. (1934).—Strabismus. London.
DOBSON, M. (1933).—Binocular Vision and Modern Treatment of Squint. London.
FRANCESCHETTI, B. (1930).—*Kurze. Handb. d. Ophthal.*, p. 685.
MAYOU, S. (1935).—*Brit. Jl. Ophthal.*, Vol. XIX, p. 37.
PARSONS, J. H. (1936).—Diseases of the Eye. 8th Edition, p. 558. London.
PETERS, A. (1924).—*Zeitschr. f. Augenheilk.*, Vol. LII, p. 369.
PETER, L. (1928).—The Extra-ocular Muscles. 1st Edition. London.
PUGH, M. A. (1936).—Squint Training. London.
RODIN, F. (1934).—*Arch. of Ophthal.*, Vol. XII, p. 874.
TRAVERS, T. A'B. (1936).—An Essay on Concomitant Strabismus. London.
WILKINSON, O. (1928).—Strabismus. Its Etiology and Treatment, p. 57.
WORTH, C. (1919).—Squint. 6th Edition. London.

I should indeed be ungrateful if first I did not acknowledge the great assistance I have received from my colleagues and friends in the compilation of this work. On this side of the Equator I extend my sincere thanks to the following:—G. H. Hogg, M.D., F.R.A.C.S., and J. L. R. Carter, M.B. (both of Launceston); W. D. Counsell, M.B.; Joseph Pearson, D.Sc.; Ralph Whishaw, M.B., M.R.C.P.; A. W. Shugg, M.B.; F. Short, B.A., LL.B., M.B.; D. H. E. Lines, M.B., F.R.A.C.S.; the late L. S. Miller, M.B.; J. Heyer, M.A.; R. S. Waring, M.A.; L. H. Payne, D.S.O.; Mrs. A. McIntyre; Miss K. Waring; Miss N. Gibson (all of Hobart); and especially to Miss L. Gilchrist (Hobart) who so skilfully drew the pedigree charts for me. On the other side of the Equator I must tender my acknowledgements to H. B. Stallard, F.R.C.S., and my most grateful thanks to the Librarian of the Royal Society of Medicine, without whose co-operation this paper could never have been written.

Secondly, I would appeal for a wider co-operation of Ophthalmologists throughout Australia, and the Empire. In Australia there is little or none. Such co-operation can only result in greater cohesion between, and greater knowledge of, the integral parts of our great Empire.

A profound ignorance of the nationality, situation, and population of Tasmania which exists throughout the world is probably mostly due to the remoteness of the island, and if this paper serves no other purpose than to bring Tasmania to the notice of Ophthalmologists throughout the Empire, then I shall be more than satisfied.

And lastly I must offer my gratitude to the Surgeons of Moorfields, whose stimulating guidance in past years has made this work possible to-day.

ACCOMMODATION IN THE EYES OF MAMMALS

BY

SIR JAMES BARRETT

MELBOURNE

THE excellent account of the theories of accommodation published by Edgar F. Fincham opens up, incidentally, the old question of whether mammals, other than primates, can accommodate at all or if so only to a negligible extent.

A great deal of experimental work has been done, and in some cases by experienced physiologists on the eyes of animals which certainly have no useful accommodation, and some erroneous conclusions have been drawn from such experimental work. Nothing is more dangerous in dealing with biological problems than the argument from analogy and also from the assumption of a supposed uniformity of type in the animal kingdom.

I venture therefore to set out briefly the history of the efforts made to ascertain what takes place in the eyes of mammals, other than primates. When the ciliary muscle was discovered by Bowman and Bruecke after 1840, and Helmholtz had in consequence developed the classical theory of accommodation, those interested (possessing the mentality of the period) assumed that the eyes of all mammals behaved just as the eyes of man did. A spate of experimental work began on the eyes of cats, dogs, and other animals which has not quite ceased yet and has sometimes, when dealing with eyes which have practically no accommodation, been misleading. But at an early date observers noted the difference between the ciliary muscle of monkeys and of cats and dogs.

The first real criticism came from Trautvetter[1] who in 1866 made a thorough investigation into the nerve control and amplitude of accommodation in the eyes of cats, dogs, rabbits and pigeons and could not find any changes in the images formed on the anterior surface of the lens by any method of stimulation of the nerves supplying the eyes in the mammals. He, however, obtained positive results in pigeons. He examined the eyes after single and double iridectomy. He repeated the work under the direction of Helmholtz with negative results. He stimulated the various nerves supplying the eyes. But it is interesting to note that even he still suggested that the animals could accommodate but that he had not discovered the method. Eserine was practically unknown for ophthalmic purposes at that date.

But in 1885, applying the then newly discovered method of retinoscopy, W. Lang and I[8] endeavoured to ascertain the refractive character of the eyes of mammals and examined 185 eyes of domestic and wild animals including rabbits, guinea-pigs, cats, rats, mice, horses and dogs. The wild animals were Siki Deer (Persia), Cervus (Porcupine), Black Bock (East Indies), Jeannette Cat (South America), Paradoxus Cat (East Indies), Peccari, Hyena, Opossum, Porcupine, Australian native cat, Jackals, Mongoose and thirteen Monkeys. In the course of this examination we found that in animals other than monkeys there was no evidence at any time of any change of refraction due to accommodation.

Theodore Beer[20] next investigated the methods of accommodation in the animal kingdom. He found that in mammals, after the primates who possess good accommodation, the others follow at a considerable interval. It attained the highest degree in animals other than primates in cats and other carnivora. As a general rule in the large eyed animals—the solipedes—the ruminants—and the whale—the range was ½ to 2D. In cats he found a range of 2D. to 3D.

But Beer makes the significant observation that in every class of animal except cephalopods and aves there are species in which accommodation is absent or is very feeble. In a letter addressed to me he stated that he found accommodation in dogs, cats and a fox. The range was 1D. to 3D., but added that it is difficult to be sure of a change of less than 2D. The errors of the method are obvious, especially to those who have examined animals' eyes by retinoscopy. Beer, however, found no accommodation in the rabbit or the hare. He found extensive accommodation (up to 10D.) in monkeys.

I[13] then iridectomised, or doubly iridectomised animals' eyes, much as Trautvetter had done, and after their recovery tested their

refraction by retinoscopy employing atropine and eserine and I checked the result by electric stimulation of the corneal margins. I failed to find any change in dogs, cats or rabbits. With monkeys there was evidence of a range of accommodation of about 5D. In the marsupials, *viz.*, the wombat and the koala—and in one monotreme (*viz.*, the echidna) there was no change. In the native cat (a marsupial) it is impossible to perform an iridectomy but there was no evidence of change. Mr. Lang and I had tested a domestic cat, thoroughly under the influence of atropine. It caught a mouse amongst obstacles without difficulty.

Treacher Collins[21] in his Bowman Lecture stated that the degree of development of the ciliary muscle corresponds fairly accurately with the degree of accommodation which the animal has been found to possess. In the echidna and the great ant eater the ciliary muscle is practically non-existent. In the rabbit only a few scanty fibres are recognisable. It is rather a commentary on the difficulty of investigation that some observers found accommodation in an animal such as the rabbit which possesses only a rudimentary ciliary muscle.

Fincham[27] has adopted a novel and ingenious method of investigation. He has photographed images on the anterior surface of the lens both in normal *situ* and after the suspension of the lens has been removed; comparing the two pictures he found that in monkeys the change was marked but in the pig, sheep, reindeer, rabbit and cat the images were slightly larger, indicating that under tension the anterior surface was slightly flatter than when the tension was removed. He estimated by retinoscopy the accommodation in a rabbit nine months old by applying atropine to one eye and eserine to the other. He found the difference was only 1D. and that the difference in the images was negligible.

Here a note of caution is necessary. Apparently eserine and atropine act as in man, but knowing the biological pitfalls I have never assumed this to be so and have checked the result by electrical stimulation at the corneal margin wherever possible.

Henderson[22] drew attention to the variations of the ciliary angle in mammals and estimated, by a method not given, the accommodation in the horse and found it not much more than 1D.

Such in brief outline is the history of this puzzling quest. Personally I found no accommodation in the eyes of the animals specified by me. Other observers found also in some instances no accommodation and in others a negligible amount. In the domestic animals Lang and I found errors of refraction amounting to 10D. of hypermetropia and also high grades of myopia and in some cases astigmatism. In the wombat I found +5D. of hypermetropia. Of what service would an accommodation amplitude of 1 to 3D. be in cases such as these?

I now set out a summary of the available knowledge of the various methods adopted in the animal kingdom as prepared by the Professor of Physiology in the University of Melbourne (Dr. W. A. Osborne)[25].

(1) In cephalopods the eyes are emmetropic or slightly hypermetropic when at rest but the lens can be pushed forward by pressure on the vitreous and a myopia of 12D. to 14D. can be produced.

(2) In marine worms (alcyopide) a bulb filled with ocular fluid and communicating with the " vitreous " is capable of being squeezed by contractible tissue and the lens is pushed forward.

(3) In the nautilus the eye is a pinhole camera and there is no lens.

(4) In fish the lens can be pulled backward in active accommodation and the eye accommodated for distant objects.

(5) In periophthalmus which must accommodate in air, the process is reversed.

(6) In amphibia the lens is drawn forward in accommodation by direct muscular action.

(7) Birds and lizards adopt the device of altering the curvature of the lens by direct pressure.

(8) In mammals the device is employed of freeing the tension on the lens and allowing its anterior surface to bulge.

It is a remarkable story and Professor Osborne's comment is apposite, *i.e.*, " the facts form a strange commentary on Sir James Jeans' belief that the architect of the universe is a higher mathematician." Perhaps some day the meaning of this puzzle may be understood.

In the meantime we may well ask how is it if there is an ascending scale of development that the primates have just learnt to do what the birds did long ago and much more successfully.

A word must be said about the gaps and failures in the animal kingdom which Beer noted in 1898. Mayou[26] found in some fresh water fishes no accommodation and apparently there is none in the sturgeon (Hess quoted by Osborne).

I referred in the commencement of this sketch to the mentality following Helmholtz' discovery of the detailed method of accommodation in man. When I prepared the paper published in the *Ophthalmic Review* in 1898 at first I submitted it to the Editor of the *Journal of Physiology* who was so sceptical of the results that he was unwilling to publish it as the conclusions were too heterodox and yet he had done experimental work on the eyes of mammals and based conclusions thereon believing that they could accommodate. Accordingly I sent it to the *Ophthalmic Review* realising that ophthalmologists would better understand the problem.

May I finally submit some questions to those interested?

(1) Of what possible value would 1D. of accommodation be to those mammals who possess it even if they could employ the whole of it—an unlikely contingency?

(2) If then it is practically useless why does the accommodation apparatus persist or re-appear?

(3) What is the meaning of the gaps and failures which seem interspersed in the animal kingdom?

(4) Why does the echidna with close reptilian affinities possess neither accommodation nor ciliary muscle, and yet the extensive amplitude in reptiles was set out by Beer and confirmed by me in the case of the Australian blue tongued lizard?

(5) How comes it that the primates have developed a method of accommodation which the birds developed long since and which is inferior to that of birds and how is it that the lower mammals have either lost it or failed to develop it? If we assume that the development of the mammals took place from a reptilian stage the problem is nearly as difficult, though the accommodation in reptiles whilst excellent is effected by methods differing from those in the birds and primates.

In conclusion, if by any flight of imagination it could be suggested that development was under anthropomorphic control it would appear that a set of diverse experiments had been made with a view to supplying these various animals with a necessary piece of apparatus and that no definite or uniform conclusion had been reached. But I prefer to state, with all humility that I cannot even conjecture how this extraordinary result has been obtained: Ignoramus and, I am afraid, Ignorabimus.

REFERENCES

1. TRAUTVETTER.—"Ueber den Nerv. der Accommodation." *Arch. f. Ophthal.*, Vol. XII (1), s. 95, 1866.
2. HENSEN and VOELCKERS.—"Experimentale Untersuchung ueber den Mechanismus der Accommodation." Kiel, 1869.
3. ADAMUK.—"Zur Frage ueber den Mechanismus der Accommodation" *Centralbl. f. d. Med. Wiss.*, p. 292, 1871.
4. *HENSON* and VOELCKERS.—"Ueber die Accommodations bewegung der Choroidea in Auge des Menschen, Affen und der Katze." *Arch. f. Ophthal.*, Vol XIX (1), s. 156, 1873.
5. J. HOCK.—"Experimentelle Untersuchungen ueber die Wirkung der Meridionalen (Langs) Fasern des Ciliar Muskels." *Centralbl. f. d. Med. Wiss.*, No. 43, 1878.
6. HIRSCHBERG.—"Zum Vergleichenden Ophthalmoscopie, Vortrag Gehalten in der Berliner Physiologischen Gesellschaft." am February 10, 1882: *Arch. f. Anat. u. Physiol.*, 1882; *Phys. Apth.*, heft 1 und 2, s. 81.
7. JESSOP.—*Proc. Roy. Soc.*, Vol. XL, p. 478, 1886.
8. LANG and BARRETT.—*Ophthal. Hosp. Repts.*, Vol. IX, p. 2, 1886.
9. HELMHOLTZ.—*Physiol. Optik*, 1866.
10. WUERDINGER.—"Ueber die Vergleichenden Anatomie des Ciliar Muskels." *Zeitschr. f. Vergleich. Augenheilk.*, s. 121, 1886.
11. LANDOLT.—"Refraction and Anomalies of the Eye." 1888.

12. JESSOP.—"The Physiology of the Intra-Ocular Muscles," Bericht Ueber die Internat. Ophthal. Congress, 1888.
13. BARRETT.—*Transactions Intercol. Med. Congress of Aust., Melbourne*, p. 588, 1889.
14. MORAT et DOYON.—*Arch. de Physiol.*, p. 507, 1861.
15. LANGLEY and ANDERSON.—"The action of Nicotine on the Ciliary Ganglion and on the endings of the Third Cranial Nerve." *Jl. of Physiol.*, Vol. XIII, p. 461, 1892.
16. ——— "On the Mechanism of the Movements of the Iris." *Jl. of Physiol.*, Vol. XIII, p. 554, 1892.
17. RICKET. Dictionnaire de Physiologie. 1895.
18. TSCHERNING.—*Optiq. Physiol.*, 1898.
19. BARRETT, J. W.—"Do Mammals Accommodate?" *Ophthal. Rev.*, Sept., 1898.
20. BEER, THEODORE.—*Wien. Klin. Wochenschr.* and *Ophthal. Rev.*, Sept., 1898.
21. COLLINS, E. TREACHER.—Bowman Lecture, 1920. *Trans. Ophthal. Soc.*, Vol. XLI. 1921.
22. HENDERSON, THOMSON.—Doyne Memorial Lecture. *Trans. Ophthal. Soc.*, Vol. XLVI, 1926.
23. WOOD, JONES F.—"The changing point of view." Bancroft Memorial Lecture. *Med. Jl. of Australia*, August 24, 1931.
24. BARRETT, J. W.—*Med. Jl. of Australia*, Feb. 6 and May 21, 1932.
25. OSBORNE, W. A.—"A Problem in Biology." *Med. Jl. of Australia*, Sept. 19, 1932.
26. MAYOU, M. S.—*Brit. Jl. of Ophthal.*, 1932 and 1933, and correspondence with J. W. BARRETT.
27. FINCHAM, EDGAR F.—"The Mechanism of Accommodation."

A MELANOMA OF THE IRIS WITH PATHOLOGICAL FINDINGS*

BY

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MELANOMATA of the iris are not of infrequent occurrence and have been recorded and exhaustively described by Fuchs, Treacher Collins, Gilbert, and Verhoeff, to mention only a few outstanding authorities. They occur, according to Fuchs, under two different forms. The first consists in a blackish tumour which apparently goes out from the stroma of the iris into the anterior chamber and arises from the proliferation of the chromatophores. The second type of melanoma has its seat at the pupillary margin of the iris and develops from the cells of the retinal pigment layer at the spot where it is reflected upon the anterior surface at the pupillary margin. Sometimes in consequence of the pupillary reaction these nodules become separated and detached and lie free in the anterior chamber. This type is not infrequently seen in the horse as has been described by Treacher Collins.

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The recognition of a pure melanoma and one as differentiated from a similar type of tumour with an underlying or complicating sarcomatous origin is frequently beset with difficulty. One is to be distinguished from the other only by determining from the previous history or from close observation, or preferably from both, whether a process of extension is in progress or not.

An instance of a pigmented neoplasm of the right eye, presumably originating from the iris stroma was brought to my attention a little over two years ago (January, 1936). From the history of its occurrence and development, if not by its actual clinical manifestations, no little concern was felt by myself as well as by the members of my staff on seeing it for the first time.

Dr. G. T., aged 24 years, consulted me on the recommendation of Dr. Evatt Mathers and Dr. Albert Doull, of Halifax, N.S., who had kept the patient under close observation for a number of years. The subject was a rather delicate looking young man, possibly due to the fact that he was just completing his final year as a student in medicine, and also because he had suffered from an attack of quinsy, for the relief of which his tonsils had been enucleated. The patient was not of the brunette type, though not a blonde, and the stroma of the irides appeared as a light grey colour. His general health had been good and there had been no tumours or instances of tuberculosis reported in his family. His physical examination was negative and no moles were present elsewhere on the body. The patient stated that a tiny pin-head pigmented spot appeared on the right iris at a point representing 10 o'clock, seven years ago. It was midway between the filtration angle and the pupillary margin. Three years ago it embraced the central half of the iris stroma, an upper and a lower segment remaining free from pigment encroachment. It was one and a half times as long as it was wide. One and a half years ago it had extended further upwards to the root of the iris, and downwards to the pupillary margin. A heaping up of the iris pigment was first apparent to his former consultants in August, 1935. The growth, apparently increasing during this space of time, became arrested and it had not extended from there until he reported to me for an opinion. The patient had never complained of pain and vision had not been impaired.

I will condense my remarks by stating that all the ocular tissues other than the iris were normal. The anterior chamber was of normal depth, the pupil was free from adhesions and active to light and accommodation. The stroma of the iris was generally of a bright grey colour. On focal examination the involved portion of the iris extended from a point just beyond 9 o'clock at the pupillary margin below to within 2 mm. of the corneo-scleral margin above, and upwards from a point representing 11.30

o'clock below at the pupillary margin up to the limitations of the root of the iris above. The area was thickened or raised, but only to a moderate degree, and not sufficiently to diminish appreciably the depth of the anterior chamber. The process of melanosis did not particularly involve the endothelial layer of the iris, and the tracery or crypts of its surface seemed to be very

FIG. 1

Radial section through iris low power, haematoxylin-eosin. Retinal pigment layer largely detached. Migration of pigment epithelium through supporting unstriped muscle cells. Pigmented portion of stroma consists of small amorphous pigment cells (chromatophores) and some definitely larger and more deeply staining cells (mesoblastic melanoblasts). Pigmented portion of stroma ceases as one approaches the pupillary margin.

little, if at all, disturbed. The slit-lamp verified these observations and also supported the opinion that the process of melanosis was sharply confined to this area, there being no pigmented extensions at either edge. There was, however, a definite ectropion of iris pigment, these cells presumably extending from the retinal pigment layer at the pupillary margin. This was a chocolate colour, while the pigmented iris above resembled more the colour of ground coffee grains. The slit-lamp did not show any excursion of pigment cells on the corneal endothelial layer in front or upon the lens capsule behind, and there were no leucocytes in the anterior chamber. There was no undue element

of vascularity on the surface of the tumour itself or near the pigmented excrescence at the pupillary margin. The pupil dilated with ease with homatropine, showing no evidence of synechiae. The fundus oculi showed no evidence of a naevus of the choroid; tension with the Schiötz's tonometer was normal;

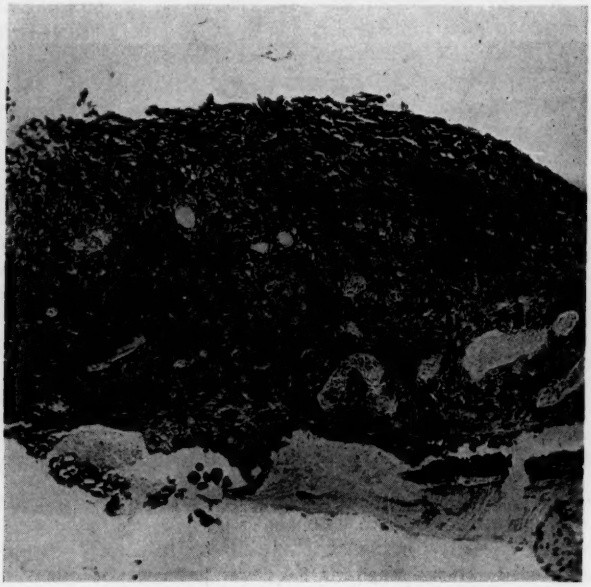

FIG. 2.

Radial section through iris, high power, haematoxylin-eosin. Iris increased at least three times its normal thickness. Little if any trace of the various component layers of iris due to the homogeneous mass of chromatophores and melanoblasts. Some apparent migration of pigment epithelium.

transillumination was negative, and vision with correction was unimpaired.

The possibly serious character presenting itself was explained to the patient, an ultimate diagnosis being reserved for a biopsy. As he was taking his final examinations a delay of four months was granted after reviewing the progress of the tumour's behaviour. On his return to Montreal, the picture was precisely the same as when he was first seen; there were no pigmentary extensions from either edge of the growth, the tumour had not increased in size, it was not engorged with blood vessels, the

pupil was active, tension was not raised, and vision with correction remained normal.

A crescentic conjunctival flap was made above to the limits of the lateral edges of the growth. An exact definition was assisted by two spots of aniline gentian violet to define the extent of the proposed corneal section. An unusually small Graefe knife was inserted just below one spot and a counter puncture made just

FIG. 3.

Radial section through iris, high power, haematoxylin-eosin. Separated pigment epithelium. Migration of pigment epithelium through unstriped muscle cells of stroma. Marked heaping up of mesoblastic melanoblasts about the walls of the dilated capillaries.

beyond the second. The incision was perilously close to the ciliary body coming out underneath the conjunctival flap. Fortunately the iris did not engage with the knife and there was no hyphaema or escape of vitreous. Tyrrell's blunt iris hook was inserted at 9 o'clock and the iris drawn out from the incision and sectioned horizontally up to its root. It was then drawn across the incision to its farthest limits and torn away from the iris root, the iris again being cut vertically in such a manner as to include completely the pigmented area in the excised portion of iris. Sutures which had been inserted as a precautionary measure were drawn up. The pillars were replaced and recovery was

uneventful. I saw the patient six months after his operation with a perfectly functioning eye. There was no further extension of pigment into the iris stroma; the upper edge of the lens, and indeed the fibres of the suspensory ligament, could be clearly seen through the coloboma with the ophthalmoscope and there did not appear to be the slightest involvement of the anterior ciliary processes. The tissue was hardened in formalin and sections cut in paraffin. These were stained in haematoxylin

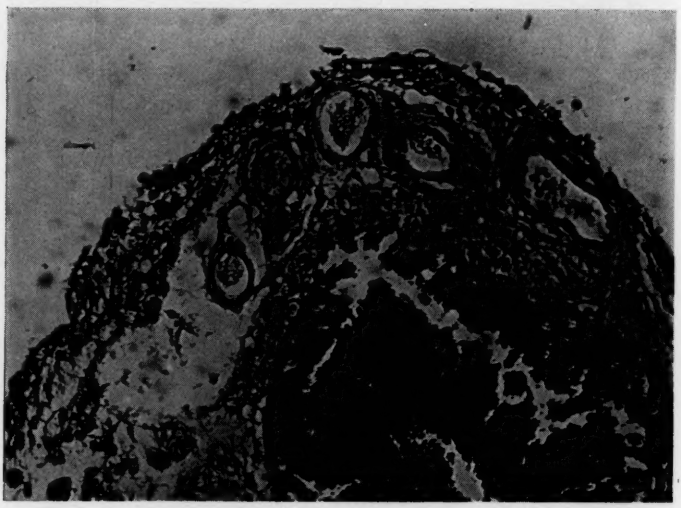

FIG. 4.

Section through pupillary margin reflecting on itself. Relatively few, if any, pigment cells within the stroma proper. Terminal capillaries of circulus iridis minor show few, if any, pigment cells about the capillary walls. No perivasculitis. Haematoxylin-eosin.

and eosin and with the same stains after bleaching. Sections were also stained with Mallory's phosphotungstic acid haematoxylin as well as by van Gieson's method.

A view of the iris on section reveals a strip of tissue apparently three times as thick as one normally finds it except at that portion where one would anticipate the presence of the sphincter iridis, namely, near the pupillary margin. On examining a section microscopically, one stained with haematoxylin and eosin, a certain definite loss of relationship has been inevitable due to a partial separation of the retinal epithelial layer in the preparation of paraffin sections. Some of these pigmented

epithelial cells are adherent and in sufficient quantity to maintain our argument. The supporting layer of unstriped muscle cells anterior to the pigment or epithelial layer, can in places be outlined with a definite element of clarity and it is in this layer of cells that a very clear picture presents itself substantiating an

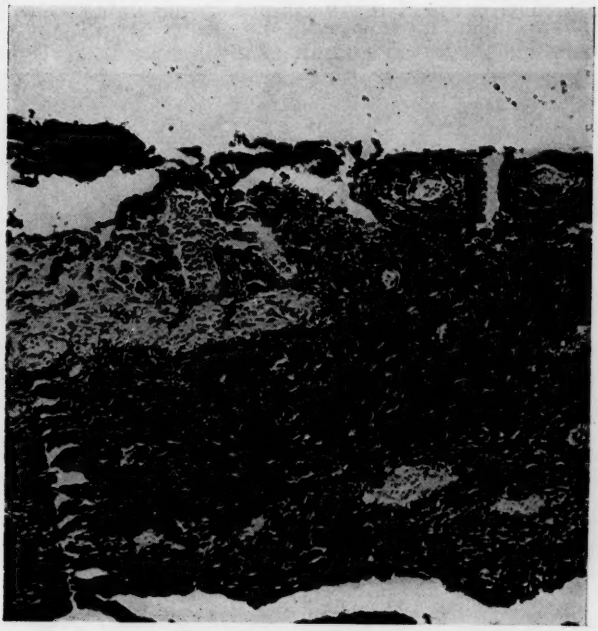

FIG. 5.

Radial section through iris, haematoxylin-eosin. Definite migration of ectodermal melanoblasts (retinal pigment epithelium) through supporting unstriped muscle tissue of stroma. These cells find their way later into the deeper layers of the iris. Many coarse pigment cells about a large capillary of the muscular layer.

idea at least regarding the genesis of the pigment cells as noted in a hyperplasia such as the case under discussion. Where unstriped muscle is present, and where the pigment layer is at least partly adjacent, one can note quite clearly a migration of these cells apparently not arrested by the muscle fibres. These pigment cells extend definitely through it and apparently into the more or less amorphous zone of the stroma proper.

A feature to be noticed during the course of migration is the affinity these cells have for the fine capillaries as one sees them in the muscular layer. The cells are, for the most part, black or chocolate colour affording little if any cell definition or distinction. Those passing through the muscle tissue are possibly sometimes smaller, possibly more definitely granular with a slightly less intense degree of pigmentation. Some of the smaller cells almost appear to have a direct relationship with the lining cells of the

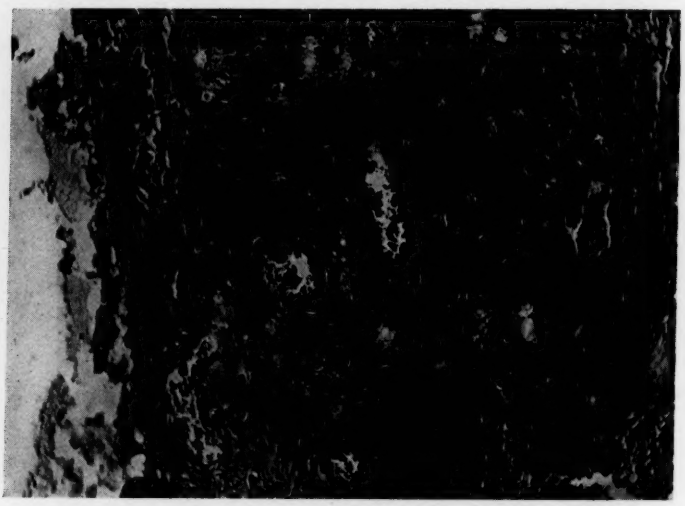

FIG. 6.

Bleached section of iris, high power, haematoxylin-eosin. Migration of pigment cells through the muscle fibres. Have largely the same characteristics of pigment epithelium, cuboid, with large nuclei. Many such cells are scattered through the stroma and others noted about the vessel walls. No signs of perivasculitis. Stroma cells proper are largely spindle cells with deeply staining nuclei. Do not branch or bud.

capillaries, while, of course, the larger ones are somewhat extrinsic or more remote. The walls of the capillaries are usually remarkably thin. Proceeding forward into the stroma proper one sees a meshwork of more or less amorphous reticular tissue supporting a conglomerate mass of cells definitely granular in character and of a light coffee ground colour as contrasted or compared with the cells of the retinal pigment layer. Interspersed within this stroma of lightly pigmented cells are scattered about larger and definitely more deeply stained cells comparable

in some respects to those of the pigment epithelium which I have already referred to as migratory cells. There seems to be a definite affinity for these larger, more deeply staining cells for the capillary walls on the part of these apparently gross feeders than is noted in that part of the iris where a hyperplasia of retinal cells is not so evident. Naturally, all definition of the endothelial cells covering the stroma is completely obscured. In bleached sections the cells of the retinal layer of the iris, or those of ectodermal origin, appear as large and epithelial in character differing from the spindle or fusiform shape of the mesodermal or melanoblastic elements of the stroma proper.

The question to me has been not so much the clinical history of the case and its subsequent behaviour following operative interference, but rather the genesis of the hyperplasia of pigment producing cells, their origin, character and the rôle they play in the formation of what some regard as a simple hyperplasia of pigment cells or others as a true tumour. The argument regarding the derivation of the cells responsible for pigmented growths has been waged by authorities of undoubted reputation, but apparently the end is not yet. The opinions advanced up to date regarding their origin are as follows:

1. They arise from the epithelium or are epiblastic in origin. (Unna, Kromeyer, Marchand, Gilchrist.)

2. They are mesoblastic in origin and are:

   a. Young connective tissue cells. (Simon, Kirchow, Riecke.)

   b. They arise from proliferation of the lymphatics. (von Recklinghausen, Lubarsch, Herxheimer.)

   c. Their origin is from the endothelium and perithelium of blood vessels. (Pick, and Jodasshon.)

   d. They originate in the sheaths of the nerve fibres. (Soldau.)

3. They are specially characterised cells of mesodermal origin. (Ribbert.)

I wish to acknowledge at the outset of my discussion the constructive assistance I have had through the co-operation and interest shown in this contribution by Dr. Theodore Waugh of the Department of Pathology, as well as that of my colleague Dr. John V. V. Nicholls, whose opinions I am happy to incorporate with my own.

The development of the iris is partly from epiderm and partly from mesoderm; each part has its own particular system of pigment cells. Those in the anterior portion of the iris, or the

mesodermal portion, are analogous to the pigmented cells of the choroid; they are generally termed " chromatophores." They, however, are more precisely termed " mesodermal melanoblasts " and correspond to the cells met with in Mongolian spots of the skin. They are " dopa " positive, at least in embryonic life and apparently may vary in their " dopa " reaction, manifesting in this way a dual function. Theories regarding the origin of these so-called mesoblastic melanoblasts are numerous. Regarding the character of this type of cell, it has been pointed out that in embryonic life they can be seen growing in from the posterior surface of the iris.

In respect to these stroma cells it would seem that light may have some effect in stimulating their activity, and it is the stimulation of such an activity, which changes the blue eyes of new-born to darker colours later in life. In their neighbourhood in the iris are histiocytes, highly phagocytic cells, which correspond to the chromatophores of the skin. These are not melanin producing cells and never " dopa " positive; they are merely reticulo-endothelial elements, branching, highly phagocytic, and store large quantities of pigment and hence are more intensely black. Whether the mesodermal melanoblasts are true mesoderm or displaced ectoderm is a debatable point; our sections at least are more suggestive regarding one point of view. Some authorities hold that these cells are simply those which have phagocytosed pigment from epithelial pigment formers; that is, that they are merely chromatophores. The " dopa " reaction would seem to rule out this possibility.

It is within the realm of physiology to have these mesodermal melanoblasts of the anterior portion of the iris, or stroma, become active pigment formers after birth, giving colour to the otherwise blue iris. It is the variations in the location and amount and character of this activity which give the marked differences in eye colour. If it is particularly emphasised in one area, definite localised dark granular spots appear in the iris. If a greater but localised or isolated area appears it is termed a " naevus or melanoma." Neither of these terms can be regarded as strictly speaking correct as used elsewhere in a pathological sense, for the cells under discussion possess no naevus cells analagous to naevi of the skin and while as a growth they might be considered tumours they are probably not neoplastic.

They may actually represent focal areas of hyperplastic activity of the mesodermal melanoblasts. I venture to suggest as a modified alternative that melanoblasts of ectodermal origin, after migration into the stroma, which represents the mesoderm, may take on altered characteristics, possibly due to their intimate association with the capillary walls. If a mass of these cells

represent an area or focus of unusual dimension probably the term melanoma is in a sense justifiable. If the reaction is more diffuse and extensive one would prefer the term " melanosis." This activity does not appear to lead to sarcoma production, though the possibility cannot be denied on pathological grounds. Such growths rarely occur from the Mongolian spots in the skin.

In contrast to the mesodermal melanoblasts we have the " ectodermal melanoblasts " which occur on the back of the iris which are an extension of the retinal epithelium and are ectodermal in origin, they are analogous to the pigment of the epidermis of the skin. Their relation to nerves as brought out by Masson is an interesting question but beyond the subject here.

It has been generally supposed that this layer of cells is much more stationary in character than the pigment cells of the stroma and is not prone to areas of hyperplasia. As I have already shown in my sections there is some definite indication, however, that cells displaced from it make their way forward where they may possibly lie in clusters known as " klumpenzellen." Their pigment is often of rod-like character and in this respect is similar to that of the posterior retinal pigment and different from the anterior uveal type. It seems quite possible, however, that they may be merely histiocytes which have phagocytosed pigment from the retinal layer—namely, true chromatophores.

Tumours of this retinal layer, as in the epidermal derivations of the skin, take the form of pigmented papillomata or warts. They frequently occur in the horse and may protrude into the pupil as Treacher Collins has shown. Some authors refer to them as melanomata or as papillary epitheliomata of the inner layer of the iris. Their cells are large and epithelial in character differing from the spindle or fusiform shape of the mesodermal melanoblastic elements. The growths are true tumours with supporting blood vessels and stroma. They lie as a rule near the pupillary border. Occasional cystic forms have been described (Gilbert). Metastasising immature growths have also been reported (Hirschberg and Birnbacher). Such, however, are best considered as melanotic carcinomata, analogous to the epidermal pigmented growths of the skin.

To complete the analogy it would seem that we should have true naevi, that is, developmental displacements of ectodermal (retinal) melanoblasts into the anterior layer. Such may occur in a sense in the previously mentioned " klumpenzellen "; but there seems to be no record of just such a case, though the present one at least has its suggestive features.

The case under discussion may come under the category of a rather extensive melanosis arising through a proliferation of the mesodermal melanoblasts until they completely fill the whole

thickness of the iris back to the posterior retinal wall. They consist of fusiform shaped cells with oval or round nuclei containing a very variable number of granules. Amongst them are larger, much more darkly pigmented, branching cells which seem more likely to be phagocytic chromatophores. The cells do not appear to be undergoing mitosis or cell division and the general character is not that of malignancy.

To emphasise the point of view concerning these cells as being constantly or even partly a contributing factor may I repeat what I have already tried to demonstrate through my sections as to the possible genesis of the stroma pigment cells. In the stroma there are two types of pigment-carrying cells concerning whose derivation there is still some confusion and doubt. It is said that they are of mesodermal origin. One of these apparently is a true chromatophore, being " dopa " negative, mesoblastic, and identical morphologically with the chromatophores of the skin. The other is " dopa " positive and it is therefore looked upon by many as a mesoblastic melanoblast. From our sections we would say rather that they are ectodermal melanoblasts as one can see them growing in from the posterior epithelial layer. Our sections would further tend to endorse the view that the physiological behaviour of these cells as noted in an embryonic state or in the process of true tumour formation is a repetition of the cellular behaviour during an embryonic state. It is certain that tumours, both functionally and morphologically are little more in some respects than a recapitulation of a more primitive or embryonic state. Von Hansemann has referred to such cell behaviour as kataplasia. This all tends to support the opinion that melanoblasts in the stroma of the iris are at least sometimes epidermal in origin. Thus the melanoma of the iris can be brought into line with Masson's theory of a nervous derivation of the skin naevi, as the ectoderm of the interior of the eye is derived from the fore-brain (diencephalon).

The problem is not simple; these mesoblastic melanoblasts of the iris stroma have the appearance of mesoblastic cells as our bleached sections show and many on very good grounds would call them mesoblastic. It is true that they are " dopa " positive (*i.e.*, they contain a specific ferment for producing melanin from dihydroxyphenylalanine) which is an ectodermal characteristic. If it is true that they are mesodermal then we have another instance, of which in the eye there are many, of a phylogenetic aberration. These are cells which partake of the characteristic of both ectoderm and mesoderm. One might mention as another instance the development of the smooth muscle of the iris from ectoderm.

REFERENCES

FUCHS.—Textbook of Ophthalmology (Duane). 1923.
——— *Arch. f. Ophthal.*, Bd. LXXXVI, S. 155.
GILBERT.—*Arch. f. Augenheilk.*, Vol. LXXXVIII, S. 143, 1921.
TREACHER COLLINS.—*Roy. Lond. Ophthal. Hosp. Reps.*, Vols. I-XII, p. 273.
COLLINS and MAYOU.—Pathology of the Eye, p. 18.
PARSONS.—Pathology of the Eye. Vol. I Part I, p. 322,
MORAX.—*Bull. de la Soc. Franç. d'Ophtal.*, Vol. XLIV, p. 365, 1931.
HOUWER.—*Trans. Ophthal. Soc. U.K.*, Vol. XLVIII, p. 167, 1928.
BOYD.—Pathology of Internal Diseases, p. 485.
MACCALLUM.—Textbook of Ophthalmology, p. 995, 1928.
MASSON —*Bull. Assoc. Franç. pour l'Etude du Cancer*, p. 303, June 20, 1921; *Ann. d'Anat. Pathol.*, Vol. III, pp. 417, 657, 1926.
VERHOEFF.—*Arch. of Ophthal.*, Vol. XL, p. 486, 1911.
SALZMANN.—Anatomy of the Human Eyeball, 1912.
HENKE and LUBARSCH.—Handbuch der speziellen pathologischen Anatomie und Histologie. Erster teil, 11/1, S. 537, 1928.

COLOBOMATA OF THE OPTIC NERVE SHEATH IN RATS

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AN examination of a collection of histological sections of rat eyes, prepared by one of us in connection with various investigations, disclosed some interesting examples of developmental defect about the optic nerve head. These were ectasias of the retina passing between the edge of the lamina cribrosa and the sclera into the optic nerve sheath, and might be considered as colobomata of the nerve sheath. Altogether sections through the optic nerve of 214 eyes were examined and most of these were serial sections. Ten cases presented this condition in greater or less degree, but showed certain differences from those described up to the present.

Quite an extensive literature has grown up around the whole subject of colobomata of the optic nerve. It is not our purpose, however, to deal with this in great detail. From our point of view it is interesting to note that Ginsberg (1896) reported a case in a rabbit showing a deep excavation of the optic nerve. A similar case in man, in which the lamina cribrosa was also defective was described by Goerlitz (1897). Von Hippel (1900) reported another, though more complicated case. Coats (1908) described two cases in an anencephalic monster where a pouch of retina ran through the lamina cribrosa into the substance of the optic nerve. In his paper, he summed up the whole question. While admitting the

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difficulties, he attempted to classify colobomata of the optic nerve under three heads: (1) where the lesion is a coloboma of the choroid beneath the nerve, the nerve itself being normal and sharing only passively in the deformity; (2) where the lesion is a coloboma of the choroid and the nerve; (3) where the lesion is a coloboma of the nerve alone, the adjacent choroid being normal. Parsons and Coats (1906) reported a variation of type (3), in that, as well as there being retina in the optic nerve, the lamina cribrosa had given way at its temporal margin where an ectasia of the retina had occurred. The scleral margin was involved also at this point. More recently van Duyse (1919) and Calhoun (1930) have described somewhat similar cases.

Kayanagi (1921) found that when rabbits are inbred, individuals are often produced which have typical coloboma of the optic nerve. He studied this problem and found that the defect is inherited as a Mendelian recessive. By intensive inbreeding he was able to produce a pure stock and so could study all stages in the development of colobomata of the optic nerve. His specimens were of the same type as ours.

It is difficult to classify our ten cases as falling in any of Coats' three types. They are similar in some respects to Parsons and Coats' case, but should, we feel, form a fourth group, judging from the position of the ectasia and the path it has followed. In three of the cases, where the condition was most advanced (Fig. 2), little or no lamina cribrosa could be seen anywhere, and in some the nerve head had been pushed back, as it were, from the eye. The retina had penetrated between the nerve and sclera on all sides, to form a sort of sleeve about the nerve within its sheath. In seven eyes the condition was much less marked (Figs. 3 and 4), the lamina cribrosa, though deficient, had separated from the sclera at only one point where the retina had pocketed through under the vaginal sheath. In two other cases the lamina cribrosa had nowhere broken away, but was weak only, and the nerve head had been pushed back a little. In no case was there either pouching of the retina into the nerve substance itself or an associated coloboma of the choroid, also the central vessels of the retina were invariably in their normal position.

We also found one specimen of a different type of coloboma of the optic nerve. For this reason it has not been included in our series. In this specimen an ectasia had taken place to one side of the nerve and thinned sclera formed the outer covering. It contained remnants of retina but no choroid. The nerve head had been slightly displaced. It belongs to type (1) of Coats' classification. We present it merely for the sake of comparison.

The essential feature, then, of the series would seem to be the poor development of the lamina cribrosa. Parsons and Coats also

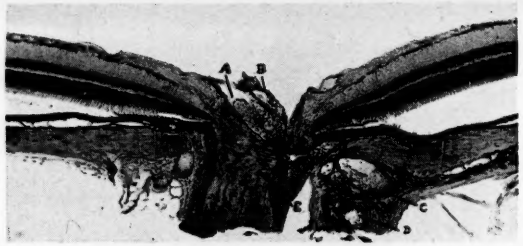

FIG. 1.

Normal optic nerve head. (A) Central vein of the optic nerve. (B) Central artery of the optic nerve. (C) Ophthalmic vein. (D) Ophthalmic artery. (E) Well developed fibres of the lamina cribrosa.

Bouin: Haematoxylin and eosin. ×52.

FIG. 2.

Section of the optic nerve surrounded by an ectasia of retina. (A) Ectasia of retina. (B) Optic nerve sheath. Note poorly developed lamina cribrosa.

Bouin: Feulgen. ×52.

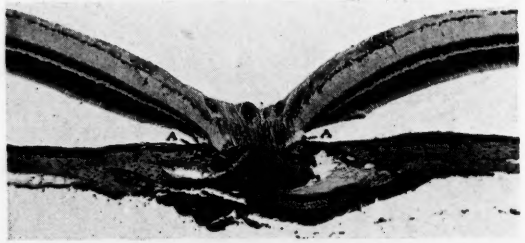

FIG. 3.

Section through optic nerve with small ectasias of retina on each side. (A) Ectasia of retina. Note poorly developed lamina cribrosa.

Bouin: Haematoxylin and eosin. ×52.

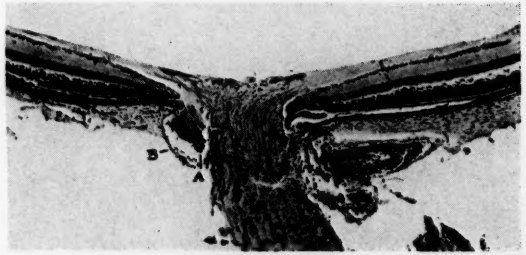

FIG. 4.

Section through optic nerve showing a small ectasia of retina (A), beneath nerve sheath (B).

Bouin: Haematoxylin and eosin. ×52.

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noticed the fragility of this structure in their case. There is some difficulty in evaluating the relationship of the ectasia to the weakness of the lamina cribrosa. Whether the ectasia follows upon the collapse of the lamina cribrosa as Parsons and Coats suggested, or both conditions are part of the same process is difficult to determine. Kayanagi's beautiful studies upon rabbits in this type of coloboma suggest the latter point of view. There is an abnormality in the condensation of the mesoderm about the neck of the primary optic vesicle so that small pouches of neural ectoderm are nipped off about the future nerve head. Because of, or along with this, there is poor development of the lamina cribrosa.

Under such circumstances it is not difficult to understand how the picture might become exaggerated as time progresses. Looked at in this way the two theories are not necessarily mutually exclusive. It also explains how in some of our cases we found the ectasia completely surrounding the optic nerve within its sheath. So far as we know this appearance has not been described before.

Coats emphasised two points in classifying colobomata of the optic nerve. Firstly, the relation of the ectasia to the intervaginal space; and secondly, the question of whether the nerve contains the central vessels. If the ectasia should approach the intervaginal space from the inner side, there must be a coloboma of the nerve; if from the outer, a coloboma of the adjacent choroid. Absence of vessels is evidence of maldevelopment, even though the nerve may have otherwise developed properly. It is also possible for the vessels to be in the normal position and yet for the cleft to be open. Thus there are exceptions to both rules. It is interesting to note, in this regard, that the vessels in all our specimens were normally placed, there was no evidence of an open nerve cleft, and the ectasia came down inside the sheath.

In one family of rats intensely inbred for the production of congenital retinal degeneration (Bourne, Campbell, and Tansley, 1938), these colobomata occurred in 5 out of 6 animals, thus showing an hereditary tendency in confirmation of Kayanagi's findings in rabbits. Those of our cases showing the most advanced condition also occurred among animals with retinal degeneration. One might be tempted to assume that there is some causal relationship between the two but the fact that colobomata of the optic nerve occur in some animals with no retinal degeneration, and that retinal degeneration usually takes place in the absence of any abnormality at the nerve head disproves this assumption.

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REFERENCES

BOURNE, M. C., CAMPBELL, D. A., and TANSLEY, K.—Hereditary degeneration of the rat retina. *Brit. Jl. Ophthal.* (in press), 1938.

CALHOUN, F. P.—Bilateral coloboma of the optic nerve. *Arch. of Ophthal.*, Vol. III, p. 71, 1930.

COATS, G.—The pathology of coloboma at the nerve entrance. *Roy. Lond. Ophthal. Hosp. Rep.*, Vol. XVII, p. 178, 1908.

GINSBERG, S.—Ueber die angeborenen Coloboma des Augapfels. *Centrabl. f. prakt. Augenheilk.*, Bd. XX, p. 225, 1896.

GOERLITZ, M.—Anatomische Untersuchung eines sogenannten Coloboma Nervi optici. *Arch. f. Augenheilk*, Bd. XXXV, p. 219, 1897.

KAYANAGI, Y.—Embryologische Untersuchungen ueber die Genese der Augenkoloboma und des Mikrophthalmus mit orbitalcyste. *Arch. f Ophthal.*, Bd. CIV, p. 1, 1921.

PARSONS, J. H., and COATS, G.—A case of orbital encephalocele with unique malformation of the brain and eye. *Brain*, Vol. XXIX, p. 209, 1906.

VAN DUYSE, G. M.—Colobomatous and Microphthalmic eyes. *Brit. Jl. Ophthal.*, Vol. III, p. 529, 1919.

VON HIPPEL, E.—*Graefe-Saemisch Handbuch des gesamt. Augenheilk.*, Pt. I, Vol. II, chap. IX, p. 27, 1900.

THE APPARATUS OF PROFESSOR ARGAÑARAZ FOR EXTRACTION OF CATARACT BY THE SUCTION METHOD (PHAKOERISIS)

THE apparatus designed by Professor Argañaraz for the operation of phakoerisis consists essentially of an ordinary glass 20 c.c. syringe (B) with its plunger (A), together with a rubber tube having moderately resistant walls (C), capable of occlusion or release by a clamp (D) and finally, the small suction cup or erisiphake (E). See Fig. 1.

The Syringe.—This is a glass syringe of 20 c.c. capacity which is capable of producing a vacuum of 5, 10 or 15 c.c., representing a negative pressure of 30, 50 or 70 mm. Hg. This vacuum can be regulated at will. If the clamp is closed and the piston withdrawn as far as the zero mark a vacuum of 15 c.c. is produced which is the longest possible with this size reservoir (A). If the plunger is only withdrawn as far as 3, 5 or 10 c.c. corresponding intensities of negative pressure can be obtained.

In order to ensure that the system is hermetically sealed, it is essential to introduce a fair amount of sterilised lanoline or other suitable fat around the piston to prevent the infiltration of any air. Metal syringes are more costly and difficult to make than glass ones. They also change their shape slightly with repeated sterilisation and are harder to replace.

The retention clip (A).—This allows the piston to be fixed in

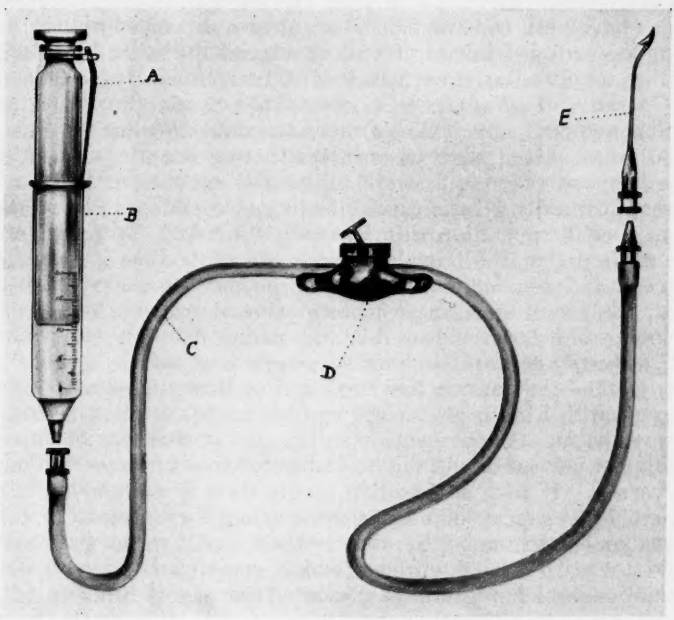


FIG. 1.

position so as to maintain the vacuum which will remain for a long time if the syringe has been smeared with lanoline. The clip (A) is fixed to a metal band around the neck of the piston and is held to this by a small screw. If the syringe breaks all that is necessary is to release the screw and the whole clip can be easily fixed to a fresh syringe.

The rubber tube.—This has a bore of 0·4 cm. and the walls are sufficiently rigid to withstand the vacuum. The tube can be boiled many times without spoiling it.

The clamp (D), which must be controlled by an assistant, is similar to those used in blood transfusions. It is only necessary to press the button of the clamp gently between the thumb and the first finger to establish direct communication between the vacuum in the syringe and the suction cup of the erisiphake. Obviously the clamp must be closed, *i.e.*, compressing the rubber tube, when the vacuum is produced in the syringe. The syringe and clamp are in the hands of the assistant during the operation.

The suction cup or erisiphake.—The cup or true pneumatic forceps fits into the rubber tube by a bulbous end. The small

spoon-like extremity is 2 mm. deep and 4-5 mm. in diameter. Two types have been constructed, in stainless metal and in silver, one rounded with a diameter of 4·5-5 mm., and the other oval similar to that used by Barraquer which is 3·5 mm. broad by 5 mm. long.

Operative Technique.—The erisiphake is not an instrument which automatically removes cataracts and one must necessarily familiarise oneself with its principle before adopting it. When one has overcome the first difficulties and has used it on 15 or 20 cases it certainly gives a greater feeling of confidence and security than does the operation with Elschnig's forceps. It must always be realised that the erisiphake is at the same time a pneumatic forceps and zonuletome. Those surgeons who are not familiar with the use of such an instrument should read the instructions below in order to avoid trouble and disillusionment.

Disadvantages of Elschnig's forceps and advantages of the erisiphake.—When one has tried two or three times to seize the capsule with Elschnig's forceps and has failed, the lens practically always becomes spontaneously subluxated so that loss of vitreous is almost inevitable and the difficulties of the extraction are much increased. If such subluxation occurs there is nothing for it but to attempt extracapsular extraction, using a cystitome, or intracapsular extraction by Smith's method. All these manoeuvres tend towards loss of vitreous which complicates or vitiates an operation, which might have succeeded completely if begun by the older methods. Many young surgeons, aware by experience of these dangers prefer to operate by the old extracapsular method which gives the greatest security and the least risk. Even in the hands of experienced surgeons the number of successful intracapsular extractions does not exceed 60 per cent. It is possible that this percentage might be improved by Arruga's technique of checking the condition of the forceps by the slit-lamp before operating. But it is an indisputable fact that when the forceps fail, which often happens, the lens is dislocated, there is loss of vitreous, cystotomy becomes difficult and in a word the use of the forceps has vitiated an extracapsular operation which might have been very satisfactory. By using a good suction apparatus all these inconveniences disappear since though it still may be difficult to luxate the lens, and the cup may slip off, if this happens the apparatus can be reapplied without difficulty or danger. The application of the Argañaraz erisiphake is made by holding the suction cup attached to the flexible rubber tube between the thumb and index finger and placing it gently on the anterior surface of the lens at the centre or anterior pole. On the other hand we should remember that the gripping of the lens by Elschnig's forceps is made completely in the dark since one cannot see the end of the forceps the surgeon does not know whether he has

succeeded in catching the lens until this is already appearing and the zonule ruptured, when the iris is pushed forwards at the moment of delivery through the pupil. In this respect the present apparatus has the big advantage that it is possible to see and control directly the point of application of the suction cup.

If the pupil has contracted during the cutting of the corneal flap it is easy to introduce the cup through the pupil and by small lateral movements to ensure that the pupillary margin is not caught by it. Thus one can avoid injuring the iris with the edge of the cup when making the vacuum. The clamp and the vacuum syringe are worked by the assistant and the surgeon need not worry about them. When he has applied the erisiphake in this way he asks the assistant to loosen the clamp to establish communication with the vacuum in the syringe so that the lens is held firmly. This is the most delicate moment of the operation since now the suspensory ligament has to be ruptured and this in young patients, under 40 years with immature cataracts may offer strong resistance.

To rupture the zonule before extraction the following procedure is to be adopted:—

1. Without exerting any pressure on the eye and by means of the suction cup the lens should be partially rotated on an antero-posterior axis, the fingers moving the applicator from one side to the other of the corneal wound. This ruptures many of the zonule fibres. 2. To complete the rupture of the remaining zonule fibres one imparts to the lens, without exerting any pressure on it, lateral " rolling " and antero-posterior " pitching " movements. In this way all the fibres of the zonule are ruptured.

3. To free the lens from all adhesions before removal it should be turned through an angle of 180° by means of the suction cup so that its posterior surface is in contact with the cornea. By continuing this movement it is extracted, forwards and upwards, that is to say through the corneal wound. In old subjects with mature cataracts the zonule is very weak and the lens can be removed from the eye without the manoeuvres mentioned above for rupturing the zonule. The younger the patient and the less mature the cataract the greater may be the resistance of the zonule and during the process of rupturing it the applicator may spring off the lens. This is not of much importance as one can take a second or even a third hold, recharging the syringe as often as is necessary to rupture the zonule and complete the extraction.

In these cases the traction on the zonule may produce a slight ciliary inflammation which shows itself as post-operative pain and delayed formation of the anterior chamber.

These symptoms, which can also follow the use of forceps are not of much importance and can be advantageously treated with fomentations and atropine.

In more than 40 patients on whom we have operated with this apparatus there has been no failure to extract the lens *in toto*.

The syringe can be charged (or rather emptied to produce the vacuum) by the assistant immediately after the surgeon has performed the section and the iridectomy, since if it is prepared at the beginning of the operation a little air may filter in and spoil the vacuum while the surgeon is performing the preliminary steps.

Iridectomy

An iridectomy should always be performed since one never knows whether the iris will prolapse the next day. Iridectomy ensures that no incarceration will occur. One can perform Hess's peripheral iridectomy or better that of Elschnig. For this the iris must be caught at the periphery and a very fine iris forceps as well as a very small de Wecker's scissors are necessary. This is followed by a reduction or smoothing out of the upper part of the pupil by an iris repositor or the blade of the de Wecker's scissors. If the mydriasis is very marked, such as is produced by subconjunctival injection of adrenalin, it is not easy to perform Hess's iridectomy. Sometimes it is almost impossible to pick up the iris in the periphery. In these cases a total iridectomy dividing the sphincter is preferable to no iridectomy at all on account of the danger of incarceration. An excellent mydriasis is usually obtained by hourly instillations of three or four drops of 2 per cent. homatropine for four or five hours before the operation. Maximum mydriasis renders the extraction easier. A drop of 1 in 1,000 adrenalin solution injected subconjunctivally near the limbus an hour before the operation gives a maximum mydriasis. Any contraction of the sphincter seriously hinders the passage of the lens through the pupil. A good mydriasis can also be obtained with glaucosan, Arruga.

The turning back of the corneal flap and the "open operation" of extraction has the disadvantage of producing a traumatic keratitis which shows itself as an opacity of the upper part of the cornea which takes several days to disappear. The corneal flap should be turned back as little as possible when doing the iridectomy. If one is doing a total iridectomy it is better to draw the iris out through the wound. The use of this simple form of erisiphake constitutes in our opinion a further improvement comparable to that marked by the introduction of Elschnig's forceps. We have no doubt that our erisiphake will supersede the forceps in the near future. Finally to obtain the best results we recommend that no departure be made from the general modern operative technique, that is to say—

1. Operate with the patient in bed using local anaesthesia (5 drops of 3 per cent. cocaine).
2. Facial akinesia, using a parotid injection of Bayer novocaine-adrenalin 5 per cent.
3. Mydriasis by homatropine (2 per cent.) 3 or 4 drops every hour, four hours before the operation.
4. Retro-ocular injection of 1 c.c. novocaine-adrenalin 1 per cent.
5. Fixation of the eye by Elschnig's suture under the superior rectus or through the episcleral tissue, as well as using the speculum, to raise the upper lid without exerting pressure.
6. Cutting a corneo-conjunctival flap and inserting a suture in this.
7. The peripheral or complete iridectomy according to degree of mydriasis taking care not to evert the corneal flap more than is necessary.
8. Without exerting any pressure above the eye apply the cup of the erisiphake smoothly to the anterior face of the lens, instructing the assistant in the meantime to release the clamp which produces the vacuum.
9. Rupture of the zonule. It is indispensable before extracting the lens to rupture the zonule by means of three movements, viz.: 1. Rotary, turning the lens on its own antero-posterior axis, displacing the handle of the erisiphake from one extremity of the wound to the other. 2. Imparting lateral rolling movements to the lens, and 3, antero-posterior pitching movements.
10. By freeing the lens from the zonule it can be extracted direct or better it can be rotated through 180° so that the posterior surface comes in contact with the cornea and is drawn out through the wound.
11. The conjunctival suture is tied and another inserted in the skin of the lid to keep the eye closed on account of the akinesia.

The Electric Vacuum Pump and the inconveniences of managing the clamp on trigger of the vacuum when this is fixed to the handle of the erisiphake

The electric motor generator invented by Barraquer to produce the vacuum required for the operation has been one of the drawbacks of the method and has prevented it becoming general. The small electric motor with its charge of oil requires constant care to ensure the satisfactory working of all the parts. Moreover the key or trigger interposed between the cup and the handle which the surgeon must release to produce communication with the vacuum, necessitates the performance of an awkward manoeuvre which makes it difficult to keep the cup gently in contact with the surface of the lens. If the two surfaces are not properly in

contact the suction of the electric motor may prove of great danger to the eye.

It is difficult for the surgeon to keep his hand steady while engaging the lens, since he must at the same time exert pressure with his finger on the trigger which puts him into communication with the vacuum. The same inconvenience is seen in the syringe vacuum apparatus of Arruga and Lijo Paoia.

Further the capacity of the syringe in this instrument was only 5 c.c., which does not seem to produce a strong enough vacuum and the frequency with which the lens slips off the cup can be put down to the feeble suction. We have seen Arruga recharge the syringe several times during the same operation.

For this reason we have adopted a syringe of 20 c.c. which can produce different degrees of vacuum between 5 and 20 c.c.

With a mature cataract and a feeble zonule in a patient of 60, 5 c.c. of vacuum is enough to extract the lens. This is equivalent to a suction of 30 mm. Hg while 15 c.c. equals 70 mm. Hg.

In certain patients, particularly those under 50 and with unripe cataracts the zonule is more resistant and a 10 c.c. vacuum is required to luxate the lens without having to reapply the cup several times while rupturing the zonule. 10 c.c. of vacuum is enough to remove the lens in most cases.

WHITE OAK HOSPITAL, 1933-37

The following report on White Oak Hospital has been submitted by Mr. Arnold Sorsby, visiting Ophthalmologist to the Hospital

The considerable administrative changes that have occurred at White Oak Hospital during the past two-and-a-half years make a comparative review of the work of the past five years desirable. The subjoined table gives the admissions for 1933-37, set out in the main groups of affections treated.

	YEAR				
Affection	*1933*	*1934*	*1935*	*1936*	*1937*
Blepharitis	69	110	134	216	260
Conjunctivitis	23	7	19	34	17
Phlyctenular Ophthalmia	54	65	93	123	163
Interstitial Keratitis	18	33	27	30	37
Trachoma	13	21	31	11	28
Miscellaneous	12	5	1	4	7
Total	189	241	305	418	512

An analysis has been made of the admissions in 1933 and 1936 as regards the duration of treatment of individual cases. Excluding cases whose treatment was not completed (owing to the children being demanded out by their parents and other reasons) the subjoined table shows the length of treatment for the more important groups treated at the hospital.

Stay at Hospital in Months	Blepharitis		Conjunctivitis		Phlyctenular Ophthalmia		Trachoma		Interstitial Keratitis	
	1933	*1936*	*1933*	*1936*	*1933*	*1936*	*1933*	*1936*	*1933*	*1936*
0— 3 -	6	46	9	8	24	11	1	1	–	2
4— 6 -	5	84	3	12	9	33	2	–	4	5
7— 9 -	3	28	5	4	5	41	–	4	2	7
10—12 -	8	7	1	–	2	8	1	1	2	7
13—18 -	8	1	–	–	2	1	3	1	4	2
19—24 -	7	–	–	–	0	–	0	–	2	–
Over 25 -	9	–	2	–	3	–	2	–	1	–
Still under treatment	–	7	–	–	–	4	–	3	–	4
	46	173	20	24	45	98	9	10	15	27

It will be seen that in 1933 of 46 cases of blepharitis fully treated, 11 or 24 per cent. were discharged within six months, whilst 15 or 32·6 per cent. required treatment for a period of one to two years and 9 or 19·5 per cent. for over two years. The figures for 1936 are a marked contrast. Of 173 no less than 130 or 75·1 per cent. were discharged within six months (46 cases or 26 per cent. being discharged within three months), whilst only one case took more than a year to cure, though to this must be added seven cases admitted in 1936 and still under treatment at the time of writing (December, 1937), making a percentage of 4·7 per cent. for cases requiring treatment for more than a year.

A further point of interest in this connection is the cases demanded out. In 1933 with a total of 189 admissions 41 were demanded, some of these cases belonging to the admissions of earlier years. Of these 41, no less than 23 were blepharitis cases, and 11 of these had been in hospital for periods ranging between 12 and 24 months, and three for 11 months. With 418 admissions in 1936, 64 children were demanded out, 38 of whom were cases of blepharitis. Of these 38 cases, 12 had been in hospital for not more than 1 month, 19 one to 3 months, six for 4 to 6 months and one for 7 months.

The figures for phlyctenular ophthalmia indicate a longer stay in 1936 than 1933. Of 45 cases treated in 1933, 24 or 53·3 per cent. were discharged within 3 months and nine or 20 per cent. within 6 months. In 1936 of 98 cases only 11 or 11·2 per cent. were discharged within 3 months, 33 cases or 33·6 per cent. within 6 months and 41 cases or 41·9 per cent. stayed at the hospital for a period of 6 to 9 months.

The figures for chronic conjunctivitis, trachoma and interstitial keratitis are too small to warrant any detailed conclusion, but generally speaking they indicate a shortening of the duration of stay. The position is therefore that in the largest group of cases, that of blepharitis, 75 per cent. of cases in 1936 as against 24 per cent. in 1933 were discharged within 6 months, and whilst 52 per cent. required treatment for more than one year in 1933, only 47 per cent. were in that position in 1936. The longer stay now given to phlyctenular ophthalmia cases was found desirable because of the recognition that these cases are potentially tuberculous. Prolongation of treatment in these cases is therefore essentially a prophylactic measure, and it would seem to have justified itself by the great diminution in the number of recurrences, as judged by the follow-up system introduced last year. Owing to the lack of a follow-up system previously, it is not possible to give comparative statistical data, but it is justifiable to assume that a diminution, in view of the fact that few recurrences are seen now, and the condition is notoriously one in which recurrences are an important and troublesome feature.

The favourable results obtained in the blepharitis groups is largely due to the introduction of treatment by antiseptic dyes instead of the time honoured methods. No small measure must also be attached to the special attention given to the general health of the children, particularly to the introduction of a richer diet. The change of diet is especially important in the satisfactory general condition of the phlyctenular group, where treatment has been essentially on general lines.

One administrative aspect deserves a special note. Whilst every precaution has always been taken at White Oak for the segregation of the infectious eye cases, children with chronic conjunctivitis were regarded as fit to mingle with others. In 1935, 78 such children were placed in a separate block of cottages and given separate classrooms of their own. As a result it was found that fewer of the other children developed acute conjunctivitis; in fact no cases of acute conjunctivitis have been observed during the past two years. The specialised attention that the group arrangement of chronic conjunctivitis cases allowed, soon brought the number of these cases down. By the beginning of 1937 it was possible to close the special block for this group of cases. Apart from the

trachoma cases—and these are rigorously segregated from the other children—and the occasional admission of a case of acute conjunctivitis—such cases being isolated till free from infection—there are now no infectious cases at White Oak Hospital.

On the clinical side, the blepharitis problem has ceased to be a major issue, but the aetiology of the affection still eludes a clear appreciation. A promising enquiry is being conducted at the Hospital by Mr. E. J. Somerset, the present holder of the Royal Eye Hospital Research Scholarship, who is studying the significance of refractive errors in blepharitis. The ample clinical material at the Hospital allowed the determination of the nature of phlyctenular ophthalmia as already noted. A final report of this subject is being completed, the preliminary report having been published in 1936. A considerable amount of work has been done on possible new treatments for trachoma and interstitial keratitis. It is hoped that a later report may be more definite on this aspect.

ANNOTATION

The Mechanism of Headache

An interesting Presidential Address, delivered before the Glasgow Northern Medical Society by Mr. J. E. Paterson, is published in the November issue of the *Glasgow Medical Journal*, and deals with some features of headache. Briefly the subject is discussed under the following headings:—experimental histamine headache; migraine; ophthalmoplegic migraine and aneurysm, and post-traumatic headaches. The author is surgeon to Stobhill Hospital and neurological surgeon to the Victoria Infirmary. He says that intra-cranial operations can readily be carried out under local anaesthesia. The bone and the brain are both insensitive; but certain parts of the dura and lepto-meninges, when stimulated, give rise to pain. It is noteworthy that such painful spots are invariably where the main vessels lie. His experiences in intra-cranial surgery lead him to the conclusion that the blood vessels of the dura and lepto-meninges conduct painful stimuli. The headache found in experiments with histamine seems to be connected in some way with the pronounced vaso-dilatation produced by the drug. Paterson agrees with the suggestion that "the headache results, not from the actual dilatation itself, but from painful stimuli from the vessel walls excited by their greatly increased amplitude of pulsation." A point stressed by the author is that "recurrent attacks of headache associated with ocular palsy nearly always, if not always, signify a leaking aneurysm of the Circle of Willis."

While there are probably many exciting causes of headache, we have always considered that a change in the intra-cranial vascular conditions was the most likely origin of the pain. So frequently patients are referred for spectacles in cases of headache; and it has always seemed to us a problem as to how many are really cured by optical appliances. Of course the error of refraction or muscular imbalance is said to act as the trigger which fires the shot. If you cut out the trigger the shot will not be fired. No doubt in a very large proportion of cases this is so; but it seems a pity that, at any rate in hospital practice, many cases are sent up for headache without much attempt having been previously made to determine if the headache is really ocular.

Over thirty years ago, when the writer was a house physician, one of his colleagues gave a headache case ten grains of aspirin on his night round. When his physician saw the drug card next day, he asked the reason; and on being informed, he called for reference cards and invited the Dental Surgeon, the Ophthalmic Surgeon, and the Nose and Throat Surgeon to see the patient, remarking to the house physician "that is the way to treat headache." The case had meanwhile recovered and the only thing that saved the Obstetrician from being consulted as well was the fact that the patient was a man. This was a case of carrying references too far. The best physician is that man who makes a thorough examination of the patient in the first instance and only applies to the special departments if his general examination reveals no obvious cause for the trouble. Headache is a marked symptom of the early stages of typhoid fever; but the man who sent a patient up with the rose spots on him with a request for spectacles would look a fool. On the other hand it does not do to treat a case as functional for too long a period, especially if an organic cause be present, without applying to the eye department. These facts should be borne in mind if we ever tend to get restive over the number of headache cases referred to us.

ABSTRACTS

MISCELLANEOUS

(1) **Hughes, Wendell L. (Hempstead). — A new method for rebuilding a lower lid.** *Arch. of Ophthal.*, June, 1937.

(1) **Hughes** describes an ingenious plastic procedure for re-making the lower lid in cases where it has been removed for a

neoplasm or as the result of trauma. The upper lid is split along the intermarginal line and the deeper layer, comprising the tarsal cartilage and conjunctiva, is pulled down and after removal of its epithelial edge is attached to the conjunctiva of the lower fornix. The skin of the cheek is undermined and mobilised so that it can be pulled up into contact with the anterior layer of the upper lid. It is then attached to the underlying tarsal cartilage of the upper lid so that the upper border of the skin occupies a transverse position mid-way between the upper and lower borders of the tarsus. The two borders of the skin are aligned by a subcuticular stitch and the lashes held upward to the skin of the upper lid by collodion. Rubber tissue is placed over the area and kept in place by a pressure bandage which is allowed to remain for six days, subsequent dressings being applied at five day intervals for three weeks. Three weeks later a row of hair is transplanted from the opposite eyebrow to the anterior surface of the tarsus just below and parallel to the upper row of lashes. Three months after the original procedure a transverse incision is made between the two rows of lashes and a complete new lower lid is then present.

F. A. W-N.

(2) **Nizetic, Zdrarko (Belgrade).—The indications and difficultles of corneal grafting.** *Klinika Oczna,* February, 1937.

(2) **Nizetic** describes his experiences of 39 cases of corneal grafting following the methods employed by Elschnig and Filatoff. The latter solved the difficulties of finding suitable donors for grafts by suggesting the use of corneal grafts from a cadaver's cornea.

Filatoff has done 95 corneal grafts in which a cadaver's cornea was used. In several cases the graft was obtained 24 hours after death.

In the author's series of 39 cases 15 grafts were taken from live donors and 24 from cadavers. In 18 of those cases the graft was obtained from the corneae of children who died from acute illnesses (enteritis, pneumonia, bronchopneumonia, asphyxia, rupture of ventricle and poisoning). In the other 6 cases the graft was taken from the corneae of adults who died as a result of accidents.

The graft is removed from the dead eye into which normal saline is injected to raise the tension. The graft is placed on gauze on a Petri dish and is covered with gauze, it is not kept in any solution and is used for grafting half-an-hour to one hour afterwards.

No difference was observed in the subsequent behaviour of the cadaver's graft when compared with the graft taken from a live donor.

The author reports improvement of vision in 10 cases out of a total series of 39. He attributes this to the fact that at first he operated on a number of unsuitable cases.

Corneal opacities following interstitial keratitis, serpiginous ulcer, eczematous keratitis are most suitable for grafting. On the other hand cases with occluded or secluded pupils, cicatrization of conjunctiva following burns and leucoma adherens are difficult and useless for grafting.

JOSEPH MINTON.

(3) **Ramsey, S. (Montreal).—Lighting as a more exact science.** *Canadian Med. Assoc. Jl.*, September, 1937.

(3) **Ramsey** comments on the fact that until recently artificial lighting for visual purposes has been almost entirely empirical. He emphasizes the importance of an accurate measurement of light to place the subject on a scientific basis. Gradually during the past 20 years more exact information has been obtained and the advent of a convenient and reasonably accurate photometer in 1931 replaced the old and cumbersome apparatus formerly in use. Since 1931 rapid progress has been achieved in the science of lighting.

Intensity of lighting for visibility only varies widely. Daylight conditions range from several hundred to about 10,000 foot candles, yet the eye can do moderately fine work such as reading under half a foot candle. But when work is done under conditions which approach the lower threshold of visibility even healthy eyes complain.

The author discusses contrasts obtaining on different papers and inks. The average book paper reflects about 80 per cent. of the incident light, while the paper of the average telephone directory, he says, reflects only 57 per cent.

Statistics compiled by the Washington Public Health Department in the New York Post Office are quoted. The eye conditions of 2,449 employees, divided between the old, poorly lighted City Hall Post Office with the more modern, better lighted offices of the City were compared. The following table gives the results:—

	City Hall	*General*
Normal vision in both eyes with no defects	10·3	19·8
Defective vision in both eyes	42·5	32·6
Refractive errors	76·6	72·5
Inflammatory conditions	20·9	11·9
Muscular imbalance...	33·4	22·4
Asthenopia	16·5	5·7

The average difference in illumination was that of 3 to 5 foot candles. These statistics are, of course, open to many errors, but the results are consistent enough to be suggestive.

Ramsey's summary, which he says is on the conservative side, is as follows:—

1. Light for reading ordinary print should be about 10 foot candles.
2. For poor print or print on paper that is not a good white 20 foot candles should be used.
3. The light should be diffused.
4. If a diffuse light is not available the intensity should be lower, about 6 foot candles, to avoid glare.
5. There should be supplementary lighting of the central field, to make it greater than the peripheral lighting.
6. No bare lights should be visible in the peripheral field.
7. Old eyes or poor eyes need more light.

R. R. J.

(4) **Bhaduri, B. N. (Calcutta).—The role of intracranial pressure in the formation of papilloedema in epidemic dropsy.** *Calcutta Med. Jl.*, August, 1937.

(4) **Bhaduri** is convinced, after the examination of the fundi in a series of nine hundred cases of epidemic dropsy, that papilloedema does exist in this condition. A paper of his in the *Calcutta Medical Journal* of August, 1935, was noticed in our columns, 1936, p. 50. In the present paper he reports another case in which it was possible to perform lumbar puncture. The patient was a woman, aged about 30 years. At her first visit the vision was R. 6/12, L. 6/18, and there was at least 2 D. of swelling of the discs. She was admitted in-patient after a fortnight's out-patient attendance. Examination of the fundi showed 4 D. of swelling on the right side and 3 D. on the left.

Lumbar puncture was done. Crystal clear fluid came out under pressure, about 3 drops per second. Nine c.c. fluid was withdrawn. The patient obtained relief from her headaches and the papilloedema rapidly diminished. The cerebro-spinal fluid showed no cellular elements and no bacteria, while the cultures were negative. The Wassermann and Kahn tests were both negative.

The author also refers to a similar case of Dr. Bose, where lumbar puncture was done with like results. He points out that the character of the cerebro-spinal fluid excludes the possibility of intracranial inflammation, and there was no clinical evidence of neoplasm.

R. R. J.

BOOK NOTICES

Text-Book of Ophthalmology. By SIR STEWART DUKE-ELDER, M.A., M.D., F.R.C.S., etc. Volume II, pp. 942, 260 Illustrations. Publishers: Henry Kimpton. Price, £3 3s. 0d.

The second volume of this admirable text-book deals with clinical methods of examination, congenital and developmental anomalies, general pathological and therapeutic considerations and diseases of the conjunctiva, cornea, and sclera.

All these are treated with a wealth of detail, and judging by the number of references quoted the author must have run the gamut of the ophthalmological literature of the world. In spite of this however, his book is no mere collection of quotations from various authors, often at variance with one another, it is an orderly and consequential whole presenting a reasoned series of facts and theories and the deductions to be drawn therefrom. Although the subjects dealt with are largely clinical in nature, the importance of their physiological and pathological aspects is always emphasized and it is this perhaps, more than any other single characteristic, which impresses the reader. Numerous examples could be adduced, a typical one occurs in the account of angular conjunctivitis where it is pointed out that the disease is really due to the disintegrating effect of a proteolytic ferment formed by a saprophytic organism, which ferment is neutralized by the anti-trypsin of the tears, and by a solution of zinc sulphate, the latter having no direct action on the diplobacillus itself.

Another striking and characteristic feature is that when the author is describing some eye condition which is part of a general disease, he also gives the features of the latter. In describing pemphigus of the conjunctiva for example, he prefaces his remarks by an account of the four different types of pemphigus of the skin, and in the description of conjunctival tularensis, by an account of the general manifestations of the disease, its mode of infection, and the history of its discovery. Such a method of treatment renders this volume more than a text-book of ophthalmology—it becomes a far wider work, which deals with those aspects of general medicine, surgery, pathology and bacteriology which have any bearing on our speciality. That the value of such an attitude is appreciated by the author himself is seen in his admirable and philosophical remarks on the general pathological and therapeutic considerations of eye disease. In the course of these he likens the eye to a "semi-isolated but co-ordinated biochemical factory, complete in itself, but at the same time working in harmony with the greater biochemical factory of the body of which it forms an integral part."

The format of the book and its general arrangement are similar

to those of volume I, and the innovation is continued in giving short biographical sketches with portraits of those who might be regarded as the patron saints of ophthalmology. The illustrations are numerous and excellent, many being in colour.

Considerations of space preclude a detailed description and appreciation of the various sections of the volume under review. Special notice must be accorded however, to the section on congenital and developmental anomalies, since these matters have never been dealt with so thoroughly outside a book devoted exclusively to their consideration. Having written this, one feels that the same remarks would apply to the other sections—in that dealing with the cornea for example, there are described no fewer than seven different varieties of keratic precipitates, in the description of conjunctival diseases, the reader is given seventeen examples of congenital and corneal diseases associated with diseases of the skin and mucous membranes and so on throughout the whole book.

In trying to sum up the qualities of Volume II, hackneyed phrases, such as "a veritable gold mine of information" come to mind, but have to be rejected because of their triteness and inadequacy, and one falls back on the simple English adjective "good." We look forward to the appearance of the remaining volumes.

Annual Report of the Department of Health of Palestine, 1936.
Jerusalem: Printing Office. Price, 130 milliemes, post free.

The Annual Report for 1936 of the Director of the Department of Public Health of Palestine, Col. Heron, has now been issued.

The estimated population of this mandated territory is about a million and a quarter. Besides ophthalmias, the endemic diseases are malaria, ankylostomiasis, bilharziasis, syphilis and tuberculosis.

The work carried out at the Ophthalmic Hospital of St. John of Jerusalem is of course the subject of a special report published by the Order. The present report, while giving some indication of the work carried out there and at the numerous other voluntary hospitals in Palestine refers principally to the Government Hospitals.

Besides the ophthalmic work at general out-patient dispensaries there are six centres at which the ophthalmic treatment is carried out in a special section of the dispensary. In addition there are two special ophthalmic clinics in Southern Palestine forming centres for the work of combating acute epidemic conjunctivitis during the summer months, and from which the temporary village first aid posts are supervised. There were 22 such centres in operation for varying periods of the year staffed by trained orderlies or nurses.

The Mobile Ophthalmic Unit carried on its work satisfactorily, and during the six months it was in the field, operated in 18 villages of the southern districts.

In contrast to the experience of 1935 the epidemic of acute conjunctivitis was this year of milder type and attended with fewer complications.

The school medical service also carried out a considerable amount of treatment of eye disease.

Viesta Ophthalmia. Volume X. Moscow, 1937.

This volume of our Russian contemporary contains many interesting papers, but none very outstanding. The Russian ophthalmic fraternity works hard, works successfully, and is a match for any of the Western European countries. There is a paper on colour sensation by Rabkin, where all modern investigating of the problem proved the excellency of the old anomaloscope of Nagel. Dashewsky constructed an ingenious little instrument to be fitted on the cornea for correct localization on the sclera of retinal lesions in need of surgical interference. Autohaemotherapy was used with indifferent results on corneal ulcers and trachomatous pannus. The impression of the reviewer is, that experiments on animals in Russia are more easily and more often performed than in this country.

The problem of post-graduate courses on ophthalmoscopy and ophthalmology is as urgent in Russia as elsewhere in the world. The most interesting are the experiments of Filatoff (Odessa) on corneal transplantation; taking the transplant from a cadaveric cornea he found that this transplantation is a powerful remedy in any inflammation of the cornea and increases all the defensive reactions of that tissue. He obtained striking improvement in cases of parenchymatous keratitis, 5 luetic and 1 tuberculous. By analogy he transplanted pieces of cadaveric skin under the foci of lupus and scleroderma and obtained striking improvement. The cadaveric skin does not disappear even two months after the operation. It is a great pleasure to notice, that in Russia, the new generation of ophthalmologists work harmoniously and in step with the general evolution of this medical branch in the world.

There is only one point for criticism. The contents of the volume are given in Russian and in German. Is it not possible to give them also in English, especially when quotations of the bibliography in individual papers are full of references to all European languages.

Detachment of the Retina and its Treatment (O Descolamento Da Retina e seu Tratramento). By BORGAS DE SOUZA (Professor of Ophthalmology in the Faculty of Medicine in Lisbon).

The author has written a short, concise and readable book on retinal detachment. He has based his work on some 500 cases that he has met with in his own consulting room and in hospital clinics, so that his statistics have been taken from all classes. These are

interesting, as unlike other authors he finds in his country that in cases of detachment the male sex does not predominate, but is practically 50 per cent. only. Among 54,681 cases he has found 0·92 per cent. of retinal detachment. He gives extensive comparison between his statistics and those of other authors, and gives statistics on spontaneous cures, which he says are rare and occur mostly in detachments secondary to choroiditis or some retinal condition, and occurs when the original condition abates. He discusses the relationship between detachment and trauma, myopia, senile decay, retinal cysts, etc., and enters at some detail into the pathology of the retina in these conditions, which predispose to detachment.

A whole chapter is given to retinal tears, their variety and cause, position and localization, pointing out that the most common position is the upper and outer quadrant of the globe, and suggesting that the traction of the superior oblique may play some part in causing the detachment. The various theories on the pathology of detachment are explained concisely, and the arguments for and against them are given very clearly and without prejudice.

In giving his statistics on post-operative cures the author points out that it is very difficult to define what is meant by a "cure" in these cases, as in some the prognosis is bad and the operation is only done to preserve some sight in the eye and as a last resource, and it is difficult to give a uniform standard of "cure." In some cases the vision is better, but the detachment may still persist and the case is not "cured," but simply better, whereas in other cases the anatomical position of the retina is perfect, but the sight does not improve and this may be due to other causes such as high myopia, vitreous haze, or that the detachment has lasted for some time and retinal degeneration has taken place.

Seventy per cent. of the author's cases had the retina in place after operation, but only about 3 per cent. had normal visual acuity (*i.e.*, 6/6), and the visual fields were normal in some 45 per cent. of cases. The operative technique is given in detail, and the author favours Lindner's operation.

This book covers the whole question of retinal detachment and its treatment, but without giving any new and original views, and it is a pity it does not contain a single diagram or illustration. The author is not, however, either dogmatic or one-sided in his views and gives the reader a very fair picture of the whole subject.

Report on The British Health Services. Quarto, pp. 430, with graphs and maps. PEP, London. Price, 7/6. 1937.

PEP, which signifies Political and Economic Planning, has done a public service in issuing this massive analysis of the Health services of Great Britain. The report is extraordinarily thorough and contains much of interest. The contents are as follows:—

Summary and conclusions; administrative machinery; impersonal protective services; industrial health; health services for mothers and children; the medical profession; other professional health services; national health insurance; institutional treatment; special services; research and genetics; nutrition; physical education; other experimental services; administrative experiments; the extent and cost of ill-health; the health problem reviewed. Two appendices are added.

Housing, water supply, sewerage, air and water pollution, noise, protection of food supply, control of the trade in medicines are a few instances of the many sided nature of the report. We note, under infectious diseases, that "vaccination has been compulsory for sixty years, but owing to loop-holes hardly more than one-third of English children are now being vaccinated."

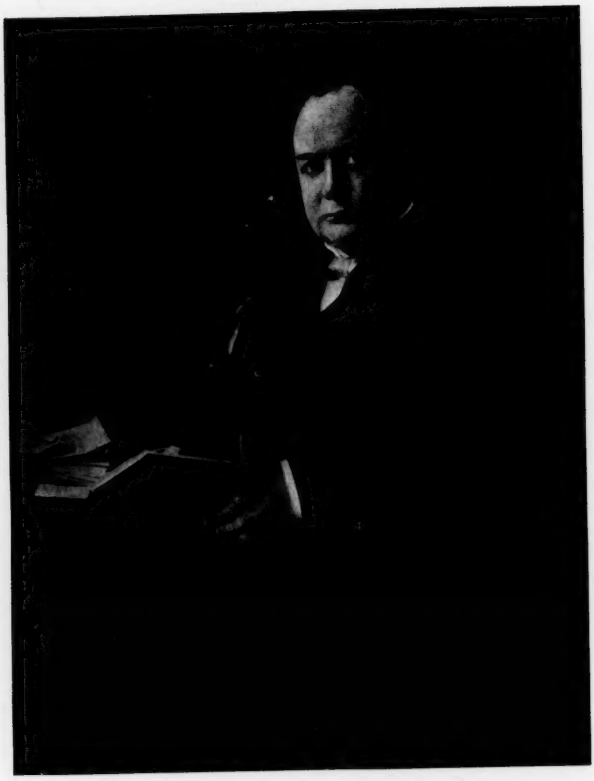
The summary of ophthalmic services states that the provision for looking after the eyes of the nation is not so satisfactory as that for the teeth of the nation. It states that the special training necessary is "not possessed by nearly enough medical men and women to meet the national requirements, and very large numbers of people have glasses, to correct their sight defects, not only made but prescribed for them by opticians, whose Joint Council has prepared a list of some 3,500 'registered optical practitioners.'"

The ophthalmic services are discussed on pages 186 to 189. We note that "about £613,000 was allocated in 1937, by societies with a membership of about 11½ millions, for ophthalmic benefit. . . . And, in 1935, schemes with a membership of 2,120,000 persons, affiliated to the British Hospitals Contributory Schemes Association, made this provision for their subscribers and their dependents."

Their conclusions are worth transcribing in full:—

"Obviously, a large section of the community needs attention to its eyes. But it may be worse than useless to receive it from unqualified men who may make gross mistakes in treating or diagnosing them. Since ocular disturbances may be a symptom of any disease, there is a danger of serious injury to a person's eyesight if a symptom which is not due to an ocular disturbance is treated as such. The situation is not analogous to that existing in regard to dentistry, since dental defect is generally a phenomenon distinct from other defects. These facts are so well known to ordinary general practitioners that they refuse to treat ocular defects unless they have had a special training.

It has been stated by the Ministry of Health that if the services of a medical eye specialist are available, any person suffering from eye trouble would be well advised to seek the services of such a specialist. Nevertheless, the public as a whole are not yet educated up to the standard of insisting on a specialist medical examination



WILLIAM GEORGE SYM

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before obtaining glasses, even where the glasses so provided would cost them no more than under an optician service. It is very doubtful if there are at the moment, or will be at any time in the near future, sufficient numbers of doctors to undertake the work. Therefore, the public will still have to be allowed to have direct access to opticians, not only to have their spectacles made but to have their eyes tested."

We were under the impression that the National Eye Service, established in 1929, is now available in more than 400 places throughout the country and has the assistance of nearly 900 specially appointed ophthalmic medical practitioners; and shall content ourselves with expressing the hope that, if the supply of trained ophthalmic specialists is not at present large enough for the needs of the Nation, it very soon will be.

Besides this, the welfare of the blind receives adequate attention on pages 302-306.

OBITUARY

WILLIAM GEORGE SYM, M.D., F.R.C.S.Ed.

We regret to announce the death of Dr. William George Sym, which occurred on January 19, 1938. Dr. Sym was born and educated in Edinburgh. His father, William Sym, was a cousin of Professor John Wilson—"Christopher North"—and a nephew of Robert Sym, W.S., the "Timothy Tickles" of the "Noctes Ambrosianae." He graduated M.B., C.M., at Edinburgh University in 1886; and M.D. in 1889, the subject of his Thesis being "Diphtheritic paralysis of accommodation and allied conditions." His interest in ophthalmology was aroused during his undergraduate career, and was related to his association with Dr. Argyll Robertson, for whom he always cherished a warm and lasting admiration.

Dr. Sym was actively connected with the Eye Department of the Royal Infirmary of Edinburgh from the year of his graduation, a connection which lasted for 34 years. He held office as Surgeon in charge of Wards from 1905 to 1920. On his resignation in 1920, the Managers appointed him Consulting Ophthalmic Surgeon to the Hospital and paid him a high tribute of appreciation for his long and much valued services.

He was a skilful operator, and in the Out-patient Department his duties were discharged with great success and acceptance. He was naturally cheerful and optimistic and his patients, while receiving the expert advice he was so well able to give, also got from him sympathy and encouragement. In his capacity as

Lecturer on Diseases of the Eye in the University of Edinburgh he proved a remarkably good teacher, clear aud arresting in his lectures, and untiring in practical exposition and clinical demonstrations. His undergraduate classes were very well attended and the students, appreciative of an enthusiastic teacher, always maintained a lively interest in the work of his class. He delighted to entertain his students with occasional amusing anecdotes which were much appreciated.

Dr. Sym started the Eye Department at Leith Hospital in February, 1896, and developed a large and increasingly busy clinic during nine years, till 1905, when he resigned. He was Ophthalmic Surgeon to the Edinburgh Eye, Ear and Throat Infirmary, where he continued his interest in clinical Ophthalmology to the end of his life. He was also for many years Ophthalmic Specialist to the Military Hospital, Scottish Command, Edinburgh.

For eleven years Dr. Sym was Editor of the *Ophthalmic Review*, which at that time, 1899 to 1909, some years prior to its incorporation in the *British Journal of Ophthalmology*, was an important Ophthalmic Journal in this country. He published a manual for students and practitioners entitled "Diseases and Injuries of the Eye," which was a useful and widely read book; and translated from the German, Adams's "Handbook of Treatment for Diseases of the Eye." He also contributed many articles on ophthalmic subjects in medical journals.

In 1911 Dr. Sym took a leading part in forming the Scottish Ophthalmological Club, and was its Secretary for twenty-two years. His active participation in discussion and his genial presence at the meetings promoted the success of the club, both in its scientific and social aspects. He was a Life Member of the Ophthalmological Society of the United Kingdom; a member of Council, 1906-1909; and Vice-President, 1918-1921. He was a Fellow of the Royal Society of Medicine; of the Royal Medical Society of Edinburgh; and of the Zoological Society of Scotland.

During the later part of his life, till within a few weeks of his death, Dr. Sym devoted his energies more exclusively to branches of the public service. He was a devoted and valued member of the Church of Scotland, and was repeatedly appointed to represent it in various capacities. He was a member of the Edinburgh Education Authority; a Governor of Donaldson's Hospital; a Director of the Royal Blind Asylum, and actively connected with many other Public Bodies.

In his wide range of reading he was especially interested in history and in biology, and took a leading part in a literary society to which he belonged. A man of robust physique, he enjoyed outdoor games, was a keen golfer and a member of the Edinburgh Medical Curling Club. He is survived by his widow, two sons and two daughters to whom our sympathy is extended in their great loss.

NOTES

Honour

SIR STEWART DUKE-ELDER has been elected an honorary member of the Royal Society of Medicine, Budapest.

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Appointments

THE following appointments have been made recently at the West London Hospital; Consulting Ophthalmic Surgeon, Mr. H. P. Gibb; Ophthalmic Surgeon: Mr. R. C. Davenport; Assistant Ophthalmic Surgeon, Mr. P. McG. Moffatt.

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Ophthalmological Society of Egypt

THE Annual Congress of the Ophthalmological Society of Egypt will take place at the Memorial Ophthalmic Laboratory, Giza, on March 25, 1938.

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Announcement of the Tenth Post-graduate Course in Ophthalmology, Vienna, 1938

THE tenth special course for post-graduate study in ophthalmology will be given between October 3 and December 7, 1938, under the auspices of the American Medical Association of Vienna at the I. and II, Eye Clinic of the Allgemeines Krankenhaus, Vienna, Austria.

This intensive post-graduate instruction was first originated in Vienna in 1922 as a result of a suggestion by Dr. Edward Jackson of Denver to Prof. E. Fuchs. Prof. J. Meller and Prof. J. Lindner, chiefs of the Eye Clinics, have again consented to take an active part. The other lectures will be given by Prof. A. Fuchs, the Docents Dr. E. Bachstez, Dr. K. Safar, Dr. L. Sallmann, Dr. F. Fischer, Dr. H. Rieger and Assistants Dr. J. Böck, Dr. W. Kreibig, Dr. F. Ramach, Dr. F. Subal and Dr. E. Pressburger, Professors of other departments will deliver lectures in their respective subjects: Prof. A. Schüller on Röntgen-rays, Prof. O. Hirsch on the Hypophysis and Sinuses; Prof. J. Bauer on Endocrine Disturbances; Prof. K. Kofler on the modified West operation.

The course has been so arranged that the field can be covered systematically and comprehensively in the allotted time. A preliminary knowledge of ophthalmology is presupposed.

Concerning operations only lectures with demonstrations will be given. In refraction only advanced work will be given.

The entire course is given in English for a minimum of ten and a maximum of seventeen men. The fee is $260 per man. Application with a certified cheque for $100 should be sent only to Prof. A. Fuchs, Vienna, VIII., Skodagasse 13. Applications are accepted

in order of priority. The application fee will be returned if the application is cancelled before September 15.

Further information can be secured by writing to Prof. Dr. A. Fuchs, Vienna, VIII., Skodagasse 13 or to the American Medical Association, Vienna, VIII., Alserstrasse 9, Café Edison.

The members meet on October 3 at 7.50 a.m. at the I. Augenklinik, IX., Alserstrasse 4, V. Hof.

FUTURE ARRANGEMENTS

1938

March 4.—North of England Ophthalmological Society, at Liverpool.

March 11.—Section of Ophthalmology, Royal Society of Medicine (Clinical Meeting).

April 1.—North of England Ophthalmological Society, at Sheffield.

April 8.—Midland Ophthalmological Society, at Nottingham Eye Infirmary.

April 28-30.—Ophthalmological Society of the United Kingdom, in London.

May 27.—Midland Ophthalmological Society, at Cheltenham Eye Hospital.

June 10.—Section of Ophthalmology, Royal Society of Medicine (Annual Meeting).

July 7-9.—Oxford Ophthalmological Congress, at Oxford.

October 7—Midland Ophthalmological Society, at Birmingham and Midland Eye Hospital (Annual Meeting).

CONTEMPORARY OPHTHALMIC LITERATURE

Archives of Ophthalmology. *January, 1938.*

GOEDBLOED. Syndrome of Groenblad and Strandberg: Angioid streaks in the fundus oculi associated with pseudoxanthoma elasticum.

ARRUGA (translated by GIFFORD). Surgical treatment of lacrymation.

MITCHELL and COOK. Galactose cataract in rats: factors influencing progressive and regressive changes.

BLOCH and GROVE. Tuberous sclerosis with retinal tumour: report of a case.

KEYES and MOORE. Adenomatous hyperplasia of the epithelium of the ciliary body: report of a case.

LAVAL. Vitamin D and myopia.

BOEDER. Power and magnification properties of contact lenses.

KENNEDY, WORTIS, REICHARD and FAIR. Adie's syndrome: report of cases.

RODIGINA. Pneumococcic bacteriophage: its application in the treatment of ulcus corneae serpens.

TERRY and FRED. Abnormal arteriovenous communication in the orbit involving the angular vein: report of a case.

NEWMAN. Diplococcus pneumoniae and streptococcus viridans in ocular diseases: report of 100 cases.
CHANG. A special form of keratitis caused by Friedländer's pneumobacillus: report of a case with review of the literature.

Annales d'Oculistique. *December, 1937.*

MAGITOT. Photophobia.
VILLARD. An oculist become pope.
GOZBERK. Palpebral manifestations of late hereditary syphilis.

Zeitschrift für Augenheilkunde. *November, 1937.*

ROLLIN. The question of the origin of plasmoma. (Pascheff.)
VON GROSZ. The skin reaction of lues from an ophthalmological point of view.
TEEGLER. Partial lipoid arcus from a tumourous process of the conjunctiva in the limbus area.
HAUSMANN. Report on short-wave ray therapy in the Universitäts-Augenklinik in Vienna.

Klinische Monatsblätter für Augenheilkunde. *November, 1937.*

JESS. Clinical tularemia especially from the ophthalmological standpoint.
DRESEL. Tularemia. Bacteriological aspect.
POLEFF. The latest result in research on the aetiology of trachoma.
DERKAC. The aetiology of trachoma.
VOLHARD. The pathogenesis of angiospastic retinitis.
GASTEIGER. Histological condition of the eyes in renal and blood pressure disorders.
KEIL. A new ophthalmodynamometer
SUGANUMA. Studies on blood pressure in the central retinal artery.
BADTKE. Calibre measurements of the retinal vessels in high pressure and renal disease.
VOGT. Dinitrophenol cataract.
BUCKLERS. Droplet shaped precipitates on the corneal surface in furniture workers
ROHRSCHNEIDER. Specific diagnosis of tuberculous eye disease.
SCHEERER. Origin of high grade myopia. Relation between excessive myopia and microphthalmos.

December, 1937.

BIETTI. Familial occurrence of retinitis punctata albescens (allied with dystrophia marginalis cristallinea corneae). Glistening of the vitreous and other degenerative ocular changes.
JANCKE. Retinal oedema as an expression of the participation of the retinal vessels in failure of the right cardiac chambers.
HAITZ. Central scotoma in congenital and squint amblyopia.
THIES. The operative treatment of ocular erosions.
STEINBERG. Constitution and ocular tuberculosis.
VON PELLATHY and NEMETH. Formations of concretions, a rarely mentioned conjunctival affliction.

Rassegna Italiana d'Ottalmologia. *September-October, 1937.*

NICOLATO. Considerations on the alterations of the optic nerve in primary glaucoma.
MALAN. Tuberculoma of the orbit.
BOSSALINO. Clinical contribution to the study of familial corneal degeneration.
PERGOLA. Social and clinical aids to the anti-trachoma struggle in the province of Sassari and results obtained in the five years 1932–1937.
PAGANI. Anaesthesia procedures in ophthalmology.
CECCHETTE. Is Credé's method for prophylaxis and cure of conjunctivitis of the newly born advisable or not?

Revista Oto-Neuro-Oftalmologica. *September-October, 1937.*

AGNELLO. Further contribution to the isolated paralysis of the abducens muscle from fracture of the base of the skull.

Anales de la Sociedad Mexicana de Oftalmologia y Oto-Rino-Laringologia. *July-August-September, 1937.*

SOLANES. Gonioscopy of surgical colobomata of the iris.
ESTRADA. Treatment of squint.
ICAZA and DUBLAN. Semeiology of the pupillary reflex.

Acta Ophthalmologica. Vol. XV, Fasc. 4, 1937.

GRANSTROM. A contribution to the knowledge of the importance of herpes infections in corneal and conjunctival infections, especially in membranous conjunctivitis.
HOLTH. Mimical ectropion or entropion of eyelids, unilateral and bilateral.
AJO and TERASKELI. Achromasia during investigation with an intense light.
PALLIN. The biorbital angle and the position of the orbital walls at different ages; their significance to the appearance and disappearance of squint.
MERKULOW. Pseudo-albuminuric retinitis of brain tumours.
HANUM. Vitreous images attending diabetic retinitis.
MAHLÉN. Treatment of severe necrosis and corrosive wounds in ophthalmology with white pellidol.
EHLERS. A thousand hemeralopia examinations.
OSTERBERG. Spasmus nutans.
SAMUELSON. A new lamp for eye examination.
NIELSEN. Convergent strabismus due to myopia.
HELMINEN. Cataract formation after atrophy with dinitro bodies.

Bollettino d'Oculistica. *November, 1937.*

CHARLIN. Nasal neuritis and trigeminal neuralgia. (Tuberculin therapy.)
ORZALESI. A case of polypus of the lacrymal sac.
MOTOLESI-BERARDI. Note on heredity in ocular diseases.
NASTRI. A case of Fehr's variegated keratitis associated with von Recklinghausen's disease.
REBUCCI. Contribution to the cure of eczematous and phlyctenular keratoconjunctivitis.
TITA. A case of unilateral esophthalmos.

Archivos de Oftalmologia de Buenos Aires. *September, 1937.*

PEYRET. Pupillometry and pharmacodynamic proof.
PAVIA. A case of direct ophthalmoscopy from a distance without an ophthalmoscope.
FORTIN. The double fovea of birds. The basic problem of visual points in relation to the anatomical topography of the retina.
VON GROLMAN. Keratoconus and trachoma.
MALBRÁN and ORIBE. Orbital mucoceles.
ADROGUÉ and RECA. A case of embolism of the superior macular artery.

October, 1937.

ARGANARAZ. The extraction of senile cataract by means of air. Vacuo-avulsion.
BALADA and FRANKS. Chiasmal alterations in arachnoiditis of the chiasma.
ZAVALIA and OLIVA. Vitreous striae of the anterior chamber.
MALBRÁN and ADROGUE. Measurement of divergent strabismus.